VOL. 18 NO. 4 OCTOBER, 1961

BRITISH JOURNAL OF INDUSTRIAL MEDICINE

EDITOR
L. G. NORMAN

ASSISTANT EDITORS
J. M. BARNES J. C. GILSON

CONTENTS

	PAGE
Rehabilitation: Concept and Practice. J. A. L. VAUGHAN JONES	241
Air Pollution in Road Tunnels. R. E. WALLER, B. T. COMMINS, and P. J. LAWTHER	250
A Clinical Study of Electrical Accidents. W. R. LEE	260
Rheumatism in Cotton Operatives. J. S. LAWRENCE	270
Further Studies on the Toxicity of Some Tetra and Trialkyl Lead Compounds. JILL E. CREMER and S. CALLAWAY	277
A Rapid Method for the Micro-estimation of Lead in Urine. J. M. DICK, R. W. ELLIS, and J. STEEL	283
The Distribution and Excretion of Inhaled Mercury Vapour. J. C. GAGE	287
The Toxicity of Precipitated Silica. P. D. BYERS and J. C. GAGE	295
Poisoning by Ethyl Mercury Toluene Sulphonanilide. M. A. JALILI and A. H. ABBASI	303
Miscellanea:	
The North of England Industrial Health Advisory Service. R. C. BROWNE and D. GRIEVES	309
General Assembly of the Permanent Commission and International Association on Industrial Health	309
Two New Medical Centres	310
Obituary	314
Book Reviews	315
Index to Volume 18	321

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

YEARLY SUBSCRIPTION (4 NUMBERS) £3. U.S.A. $10.00 SINGLE NUMBER 17/6

EDITORIAL COMMITTEE

THOMAS BEDFORD
SIR ROY CAMERON
C. M. FLETCHER
M. W. GOLDBLATT
A. G. HEPPLESTON
ALASTAIR HERON
I. T. T. HIGGINS
A. BRADFORD HILL
T. G. FAULKNER HUDSON
R. E. LANE
J. F. LOUTIT
H. B. MAITLAND
A. MEIKLEJOHN
J. N. MORRIS
P. A. B. RAFFLE
R. S. F. SCHILLING
J. A. SMILEY
J. R. SQUIRE

EDITOR, *British Medical Journal*

GENERAL ADVISORY BOARD

A. J. AMOR
SIR FREDERICK BARTLETT
SIR HENRY BASHFORD
G. P. CROWDEN
DONALD HUNTER
SIR WILSON JAMESON
T. A. LLOYD-DAVIES
J. M. MACKINTOSH
E. R. A. MEREWETHER
D. C. NORRIS
H. OSMOND-CLARKE
K. M. A. PERRY
DONALD STEWART
SIR REGINALD WATSON-JONES

President of the Association of Industrial Medical Officers and Hon. Editor of the Transactions of the Association of Industrial Medical Officers, *ex officio*

NOTICE TO CONTRIBUTORS

The *British Journal of Industrial Medicine* is intended for the publication of original contributions in industrial medicine from workers of any nationality. It also provides a section for book reviews.

All papers submitted for publication should be referred to The Editor, British Journal of Industrial Medicine, Griffith House, 280 Marylebone Road, London, N.W.1.

Papers are accepted on the understanding that they are contributed solely to this Journal, and that they are subject to editorial revision. Papers should be prefaced by an abstract of the argument and findings and should be more comprehensive than a summary, which will not be required. Papers must be typewritten on one side of the paper only, in double spacing, and with a margin of at least 1½ in. Photographs and photomicrographs should be printed on glossy paper, and should be unmarked. Charts and graphs accompanying papers should be carefully drawn in black ink on tracing linen or Bristol board or stout, smooth, white paper. Any lettering on these drawings to be done in the editorial office should be lightly inserted in pencil.

References should be arranged according to the Harvard system. When a book is referred to, the place and year of publication, edition, and page should be given. In the text the year of publication must follow the author's name, more than one paper in any one year being indicated by a small letter (a, b, c) after the date. No numbering of references is necessary.

Contributors will receive one proof, but it is assumed that all but verbal corrections have been made in the original manuscript; an allowance at the rate of 10s. per sheet of 16 pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

Fifty free reprints of articles will, if desired, be given to contributors. A limited number of additional reprints at cost price can be supplied if application is made when returning proofs. An estimate of costs will be given on application to the Publishing Manager, British Medical Association.

Papers which have been published become the property of the *British Journal of Industrial Medicine* and permission to republish must be obtained from the Editor.

Application for advertisement space should be addressed to the Advertisement Manager, British Medical Association, Tavistock Square, London, W.C.1.

NOTICE TO SUBSCRIBERS

Subscriptions are payable to the British Medical Association. Address: British Medical Association House, Tavistock Square, London, W.C.1.

IN CRISIS AND CONVALESCENCE

COMPLAN provides *complete* nourishment

Complan

Contains every nutrient essential to life

GLAXO

EDITORIAL COMMITTEE

THOMAS BEDFORD
SIR ROY CAMERON
C. M. FLETCHER
M. W. GOLDBLATT
A. G. HEPPLESTON
ALASTAIR HERON
I. T. T. HIGGINS
A. BRADFORD HILL
T. G. FAULKNER HUDSON
R. E. LANE
J. F. LOUTIT
H. B. MAITLAND
A. MEIKLEJOHN
J. N. MORRIS
P. A. B. RAFFLE
R. S. F. SCHILLING
J. A. SMILEY
J. R. SQUIRE

EDITOR, *British Medical Journal*

GENERAL ADVISORY BOARD

A. J. AMOR
SIR FREDERICK BARTLETT
SIR HENRY BASHFORD
G. P. CROWDEN
DONALD HUNTER
SIR WILSON JAMESON
T. A. LLOYD-DAVIES
J. M. MACKINTOSH
E. R. A. MEREWETHER
D. C. NORRIS
H. OSMOND-CLARKE
K. M. A. PERRY
DONALD STEWART
SIR REGINALD WATSON-JONES

President of the Association of Industrial Medical Officers and Hon. Editor of the Transactions of the Association of Industrial Medical Officers, *ex officio*

NOTICE TO CONTRIBUTORS

The *British Journal of Industrial Medicine* is intended for the publication of original contributions in industrial medicine from workers of any nationality. It also provides a section for book reviews.

All papers submitted for publication should be referred to The Editor, British Journal of Industrial Medicine, Griffith House, 280 Marylebone Road, London, N.W.1.

Papers are accepted on the understanding that they are contributed solely to this Journal, and that they are subject to editorial revision. Papers should be prefaced by an abstract of the argument and findings and should be more comprehensive than a summary, which will not be required. Papers must be typewritten on one side of the paper only, in double spacing, and with a margin of at least 1½ in. Photographs and photomicrographs should be printed on glossy paper, and should be unmarked. Charts and graphs accompanying papers should be carefully drawn in black ink on tracing linen or Bristol board or stout, smooth, white paper. Any lettering on these drawings to be done in the editorial office should be lightly inserted in pencil.

References should be arranged according to the Harvard system. When a book is referred to, the place and year of publication, edition, and page should be given. In the text the year of publication must follow the author's name, more than one paper in any one year being indicated by a small letter (a, b, c) after the date. No numbering of references is necessary.

Contributors will receive one proof, but it is assumed that all but verbal corrections have been made in the original manuscript; an allowance at the rate of 10s. per sheet of 16 pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

Fifty free reprints of articles will, if desired, be given to contributors. A limited number of additional reprints at cost price can be supplied if application is made when returning proofs. An estimate of costs will be given on application to the Publishing Manager, British Medical Association.

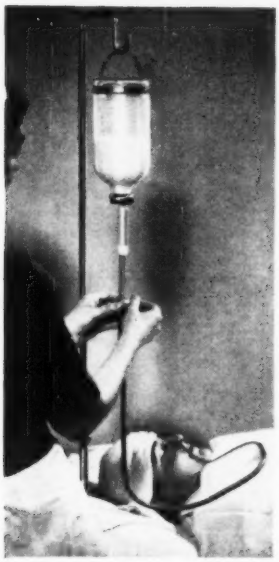
Papers which have been published become the property of the *British Journal of Industrial Medicine* and permission to republish must be obtained from the Editor.

Application for advertisement space should be addressed to the Advertisement Manager, British Medical Association, Tavistock Square, London, W.C.1.

NOTICE TO SUBSCRIBERS

Subscriptions are payable to the British Medical Association. Address: British Medical Association House, Tavistock Square, London, W.C.1.

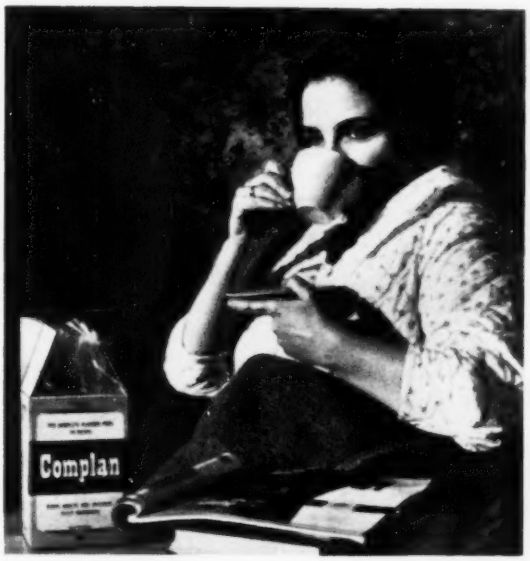
IN CRISIS AND CONVALESCENCE



COMPLAN provides *complete* nourishment

The same Complan which is so widely used in hospital is so beneficial when patients return home. Two or three cups daily of Complan (so very easily prepared) provide the high protein-vitamin-mineral intake to build them up to full health.

And it's not just recently hospitalised patients who make Complan customers . . . children with mumps or tonsilitis . . . gastric sufferers who need a bland diet and old people who subsist on meagre meals—these are just a few of the many who buy Complan and come back for more.



Complan

GLAXO

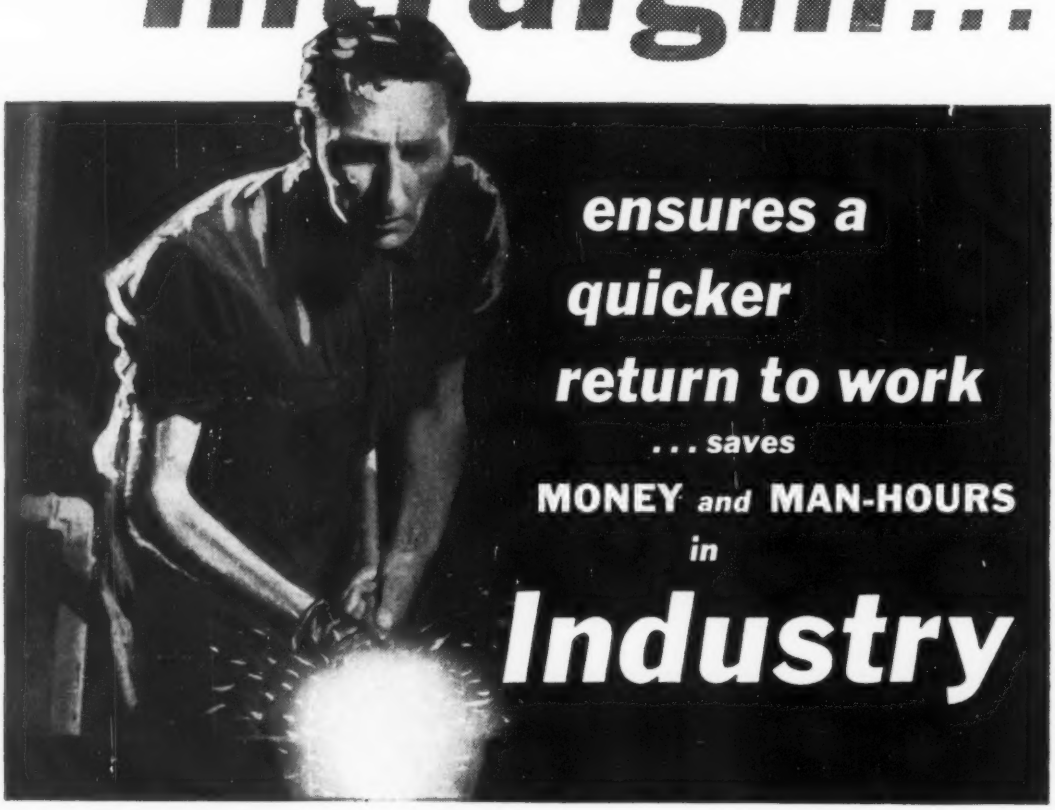
COMPLAN *is a Glaxo Trade Mark*

Contains every nutrient essential to life

GLAXO LABORATORIES LIMITED,
GREENFORD, MIDDLESEX, BYRon 3434
Subsidiary companies or Agents in most countries

In MUSCLE INJURY

Intralgin...



to apply **Intralgin Liquid**
(8 & 32 oz. bottles ... 6/8 & 19/8) bandage
a wet dressing in position

... to apply **Intralgin Gel**
(50 gramme tube ... 3/4) use
liberal amount and gently
rub in
... for a more intense effect
—warm area first

Samples and literature are available on request

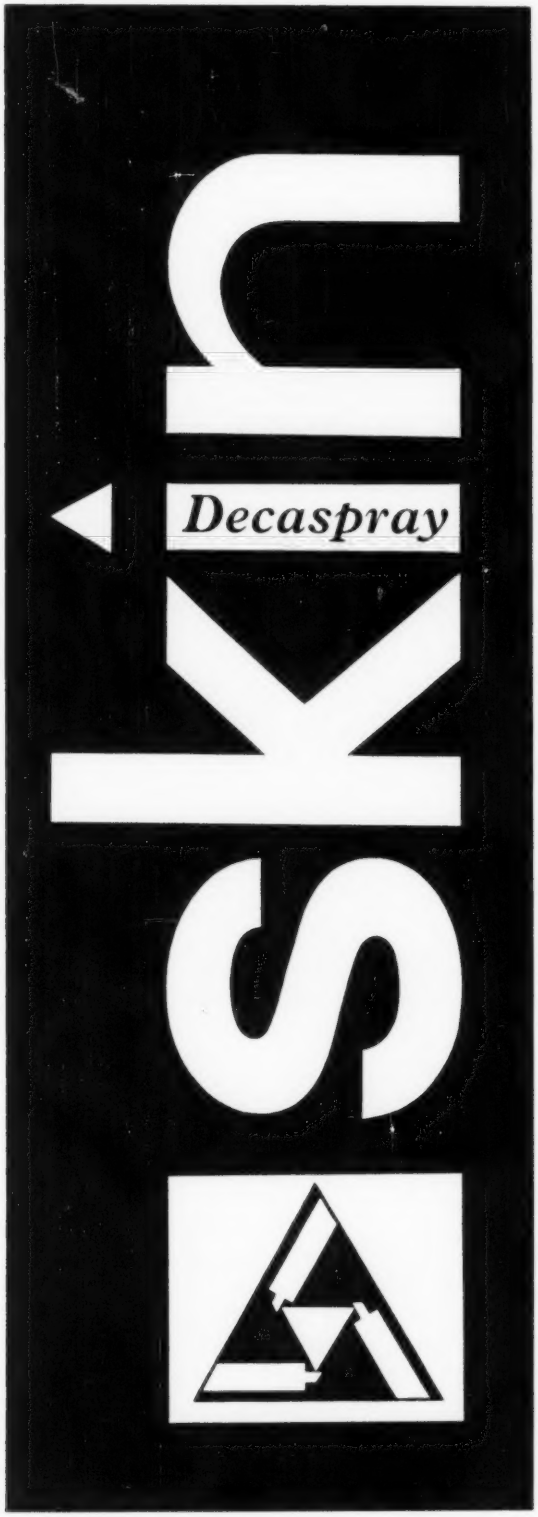
Intralgin applied externally
rapidly relieves muscular pain

Intralgin combines
BENZOCAINE and SALICYLAMIDE
which penetrate the skin and act locally in muscles
to relieve pain resulting from
SPRAINS ... STRAINS ... and INJURIES
—by crush or impact.

RIKER

'Intralgin' is a registered trade mark of

RIKER LABORATORIES LIMITED LOUGHBOROUGH LEICESTERSHIRE
In-6-UK



—a marked advance in the treatment of skin disease

'DECASPRAY' represents a striking advance on other methods; it offers unexcelled convenience, uniformity of application, and reduced risk of spreading dermatoses and introducing infection. The cooling propellant promptly relieves burning and itching. From every angle 'DECASPRAY' is the modern treatment for skin disease.

Decaspray* TOPICAL AEROSOL

DEXAMETHASONE and NEOMYCIN SULPHATE

Precautions: 'DECASPRAY' should not be used in the presence of tuberculosis of the skin. It should not be sprayed into the eyes, ears or nose.

Supplied in 90 G. spray pack, containing 10mg. dexamethasone and 50mg. neomycin sulphate, sufficient for approximately 130 separate applications. Also available as 'DECASPRAY' small, in 30 G. spray pack.

MSD MERCK SHARP & DOHME LIMITED HODDESDON, HERTS

* Trademark

A BACTERICIDAL DRESSING

which ensures rapid
sterilisation of
infected wounds

Sofra-Tulle

Paraffin Gauze Dressing with 1% of Soframycin

—provides excellent protection
for the lesion
—does not macerate
—does not sensitise
—is well tolerated

Packs: 10 pieces 4"×4"
Strip..... 4"×40"

ROUSSEL LABORATORIES LTD.
LONDON, N.W.10
LADbroke 6611

ROUSSEL

DEEP SKIN PENETRATION IN ARTHRITIS AND RHEUMATISM

NICOTINIC, SALICYLIC AND p-AMINOBENZOIC ACIDS in Ester form

IN CASES of soft-tissue rheumatism, and arthritic disorders, many doctors are tending more and more to regard Transvasin as an indispensable adjuvant to treatment.

For Transvasin is composed of the esters of nicotinic, salicylic and p-aminobenzoic acids. These esters readily pass the skin barrier in therapeutic quantities, and so enable an effective concentration of drugs to be built up where they are needed.*

Transvasin not only induces vasodilatation of the skin with a superficial erythema, but also brings about deep hyperaemia of the underlying tissues. It is non-irritant and can be safely used on delicate skins.

It is now being widely prescribed, with successful clinical results. Since a very small quantity is sufficient for each application, the cost of treatment is extremely low. Transvasin can be prescribed on Form EC10.

**Therapeutische Umschau 1952, 8, 143*

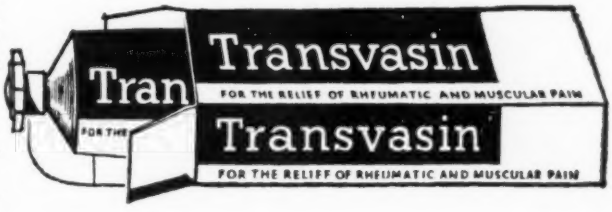
"Thank you, doctor"

LLOYD-HAMOL LIMITED

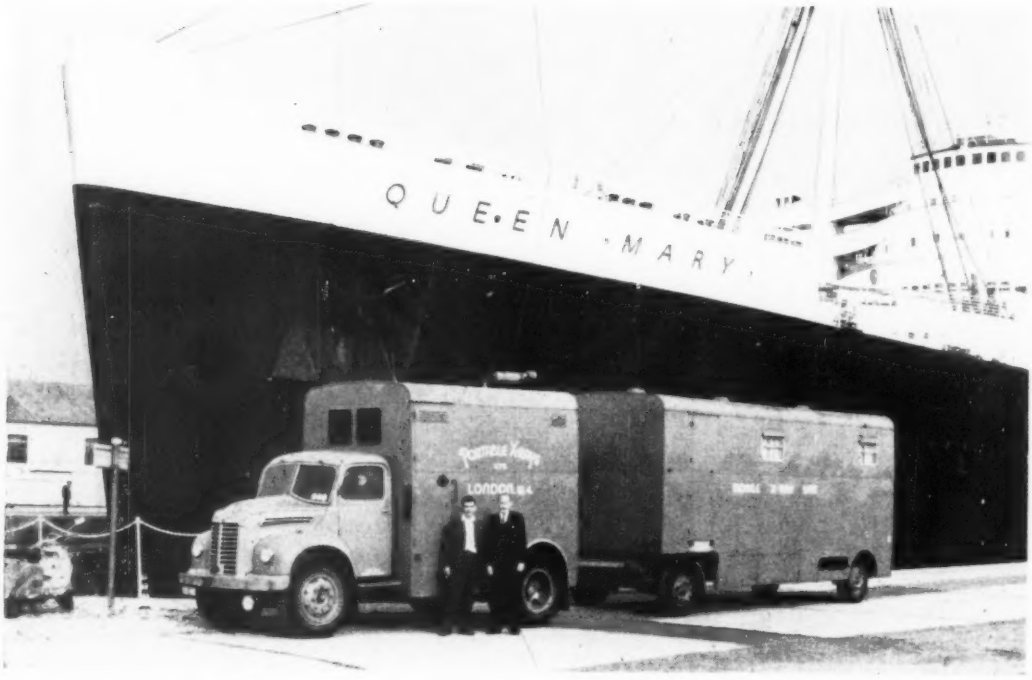
11 Waterloo Place, London, S.W.1 Telephone WHitehall 8654/5/6

Transvasin is available in 30g. tubes, N.H.S. price in the U.K. 2/6 plus P.T., and is not advertised to the public. Samples and literature will be gladly sent on application.

Tetrahydrofurfuryl salicylate	14%
Ethyl nicotinate	2%
n-Hexyl nicotinate	2%
Ethyl p-aminobenzoate	2%
Water-miscible cream base ad	100%



Don't waste man hours -it's costly!



X-Ray Unit in position ready to commence examinations

Please write stating your requirements for quotation or ask our representative to call.

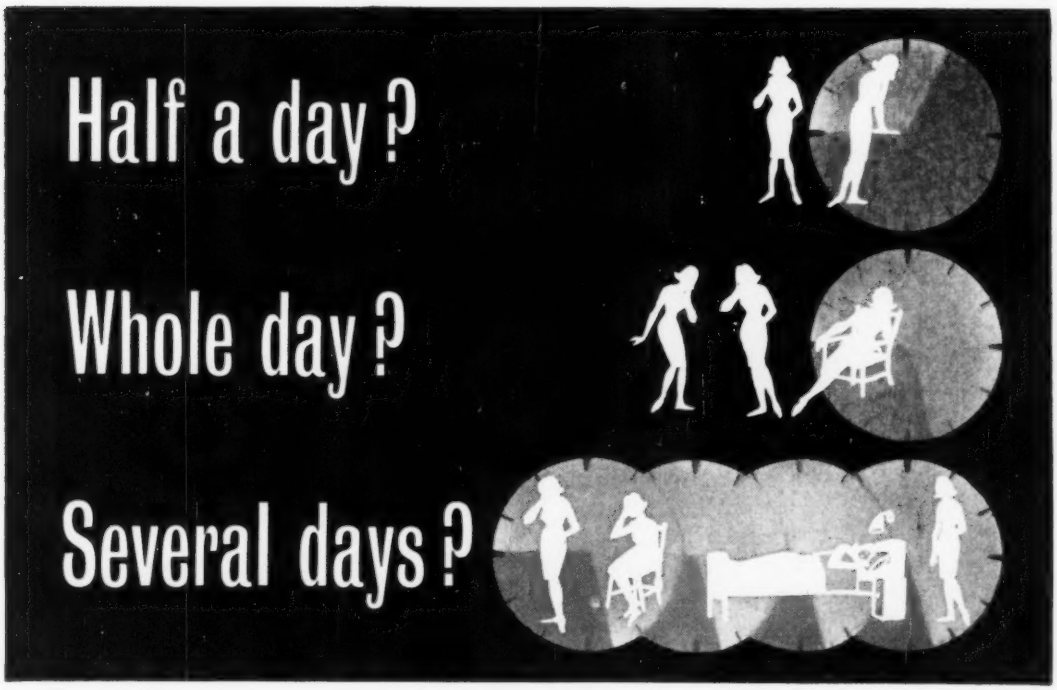
To keep the 'Queen of the Seas' in dock is a costly business. That is why Portable X-Rays were chosen by the London Hospital Department for Research in Industrial Medicine (Medical Research Council) to assist in a special investigation into the health of the Boiler Scalers on the 'Queen Mary'.

Mobile X-Ray Units of Portable X-Rays Ltd. are regularly employed by large industrial organisations for chest examinations on site during working hours with economy in time and money.

PORTABLE X-RAYS LIMITED

POWER ROAD, CHISWICK, LONDON, W.4. *Tel. : CHIswick 7836/7*

STAFF SICKNESS.....



Control of two major factors in staff sickness

Gastric disturbances in both men and women, and **Primary Dysmenorrhoea** among female staff probably account for a relatively high proportion of 'sick leave' or sub-standard effort in the majority of industrial organisations.

BISLUMINA

The antipeptic-antacid Bislumina, containing bismuth aluminate in combination with magnesium oxide, embodies all those properties considered desirable in an agent for the local treatment of peptic ulcer and allied dyspeptic disorders.
Available as tablets in packs of 12, 30, and 250.

FEMERITAL

A specific uterine spasmolytic plus effective analgesic action provides reliable therapy for primary dysmenorrhoea. Femerital contains dibutamide together with phenacetin, salicylamide and caffein. Available in boxes of 30 tablets and packs of 504 tablets.

Further information and samples available on request.

M.C.P. Pure Drugs Ltd. STATION WHARF, ALPERTON, MIDDLESEX. Tel: WEMbley 1191.

5 drops for the removal of EAR WAX

Cerumol avoids discomfort to the patient, inconvenience to the doctor and saves time for both.
Cerumol is now routinely used in most hospitals and general practices throughout the country.
Satisfactory results confirm that it is the most efficient agent yet introduced for the purpose and that it is exceptionally reliable and safe.
In 10-30 minutes the softened or disimpacted wax can usually be wiped out with a probe tipped with cotton wool, or by gentle syringing.
Patients can readily be instructed to instil the drops themselves and in many cases the loosened wax will run out of its own accord.

sterilisation of

infected wounds

Sofra-Tulle

Paraffin Gauze Dressing with 1% of Soframycin

—provides excellent protection for the lesion
—does not macerate
—does not sensitise
—is well tolerated

Packs: 10 pieces 4" × 4"
Strip..... 4" × 40"

ROUSSEL LABORATORIES LTD.

LONDON, N.W.10

LADbroke 6611

ROUSSEL

dramatic

FOR **SUB-ACUTE** and chronic dermatoses

TAR

+

HYDROCORTISONE

combined in

ARTHRITIS AND RHEUMATISM

NICOTINIC, SALICYLIC AND p-AMINOBENZOIC ACIDS in Ester form

IN CASES of soft-tissue rheumatism and arthritic disorders many doctors are tending more and more to regard Transvasin as an indispensable adjuvant to treatment.

For Transvasin is composed of the esters of nicotinic, salicylic and p-aminobenzoic acids. These esters readily pass the skin barrier in therapeutic quantities, and so enable an effective concentration of drugs to be built up where they are needed.*

Transvasin not only induces vasodilatation of the skin with a superficial erythema, but also brings about deep hyperaemia of the underlying tissues. It is non-irritant and can be safely used on delicate skins.

It is now being widely prescribed, with successful clinical results. Since a very small quantity is sufficient for each application, the cost of treatment is extremely low. Transvasin can be prescribed on Form EC10.

**Therapeutische Umschau 1952, 8, 143*

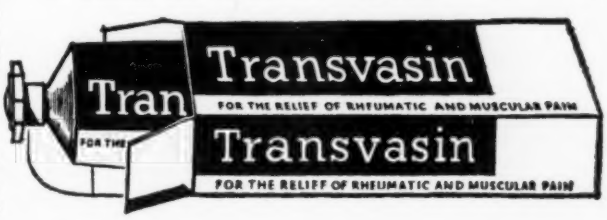
"Thank you, doctor"

LLOYD-HAMOL LIMITED

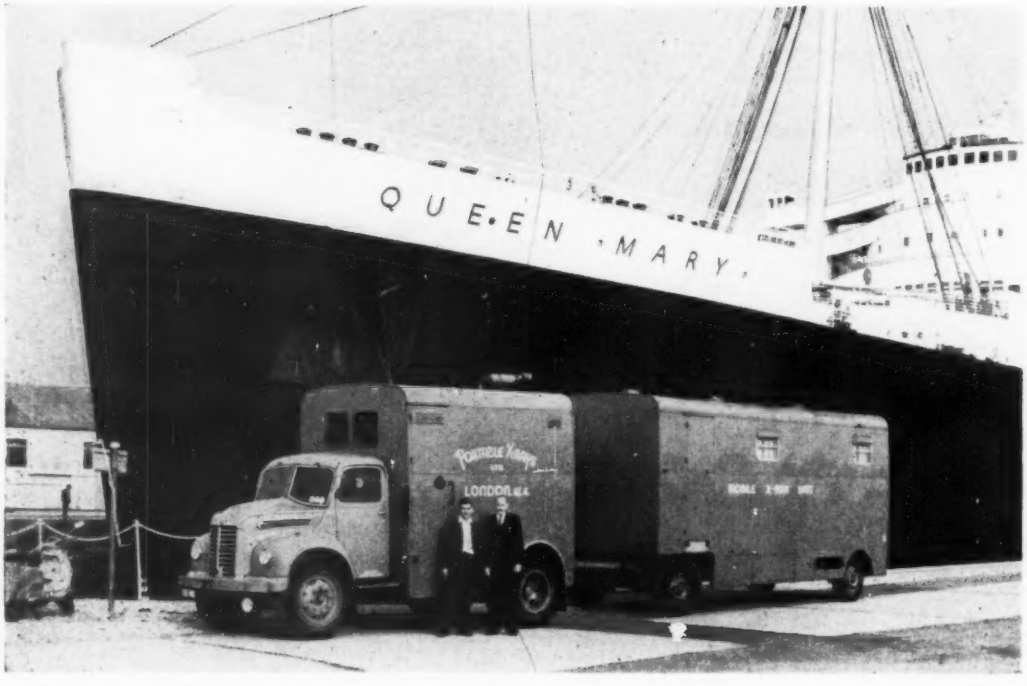
11 Waterloo Place, London, S.W.1 Telephone WHitehall 8654/5/6

Transvasin is available in 30g. tubes, N.H.S. price in the U.K. 2/6 plus P.T., and is not advertised to the public. Samples and literature will be gladly sent on application.

Tetrahydrofurfuryl salicylate	14%
Ethyl nicotinate	2%
n-Hexyl nicotinate	2%
Ethyl p-aminobenzoate	2%
Water-miscible cream base ad	100%



Don't waste man hours -it's costly!



X-Ray Unit in position ready to commence examinations

Please write stating your requirements for quotation or ask our representative to call.

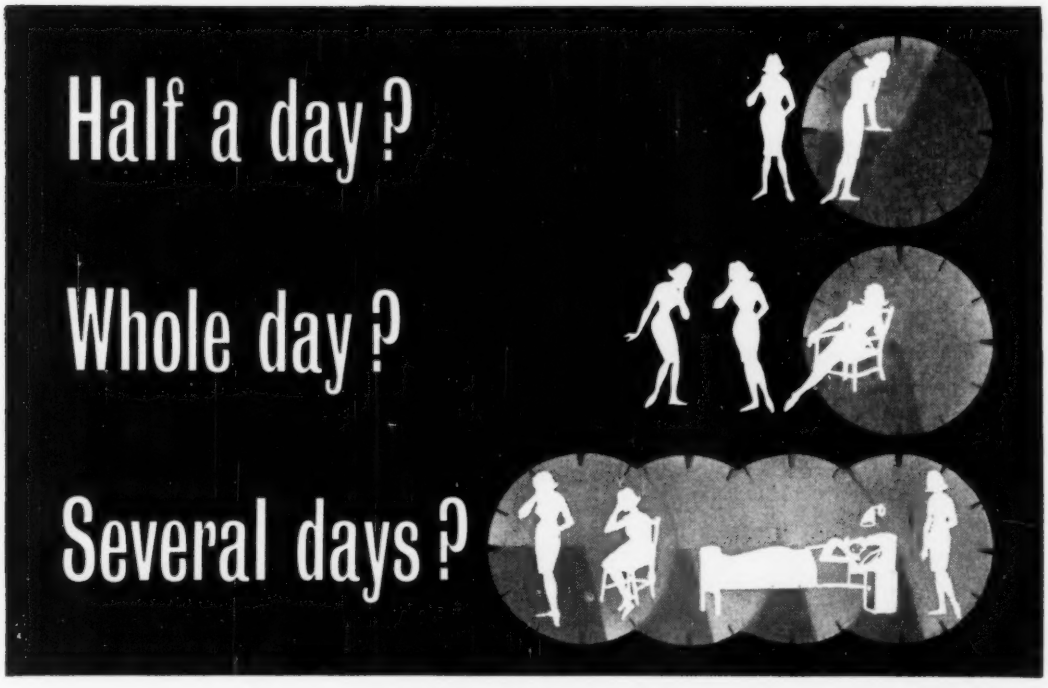
To keep the 'Queen of the Seas' in dock is a costly business. That is why Portable X-Rays were chosen by the London Hospital Department for Research in Industrial Medicine (Medical Research Council) to assist in a special investigation into the health of the Boiler Scalers on the 'Queen Mary'.

Mobile X-Ray Units of Portable X-Rays Ltd. are regularly employed by large industrial organisations for chest examinations on site during working hours with economy in time and money.

PORTABLE X-RAYS LIMITED

POWER ROAD, CHISWICK, LONDON, W.4. *Tel. : CHIswick 7836/7*

STAFF SICKNESS.....



Control of two major factors in staff sickness

Gastric disturbances in both men and women, and **Primary Dysmenorrhoea** among female staff probably account for a relatively high proportion of 'sick leave' or sub-standard effort in the majority of industrial organisations.

BISLUMINA

The antipeptic-antacid Bislumina, containing bismuth aluminate in combination with magnesium oxide, embodies all those properties considered desirable in an agent for the local treatment of peptic ulcer and allied dyspeptic disorders.
Available as tablets in packs of 12, 30, and 250.

FEMERITAL

A specific uterine spasmolytic plus effective analgesic action provides reliable therapy for primary dysmenorrhoea. Femerital contains dibutamide together with phenacetin, salicylamide and caffein. Available in boxes of 30 tablets and packs of 504 tablets.

Further information and samples available on request.

M.C.P. Pure Drugs Ltd. STATION WHARF, ALPERTON, MIDDLESEX. Tel: WEMbley 1191.

5 drops for the removal of EAR WAX

Cerumol avoids discomfort to the patient, inconvenience to the doctor and saves time for both.
Cerumol is now routinely used in most hospitals and general practices throughout the country.
Satisfactory results confirm that it is the most efficient agent yet introduced for the purpose and that it is exceptionally reliable and safe.
In 10-30 minutes the softened or disimpacted wax can usually be wiped out with a probe tipped with cotton wool, or by gentle syringing.
Patients can readily be instructed to instil the drops themselves and, in many cases, the loosened wax will run out of its own accord, rendering further attendance at the surgery unnecessary.

CERUMOL
REGD. TRADE MARK

Active constituents per 100 ml.

p-dichlorbenzene B.P.C.	**2 G.**
Benzocaine B.P.	**2.7 G.**
Chlorbutol B.P.	**5 G.**
Ol. Terebinth B.P.	**10 ml.**

Pack for surgery use—dropper vial. Basic N.H.S. Cost 2/8.
Hospital Pack—2 oz. bottles.

STABILIZED KOLA AND VITAMIN TONIC

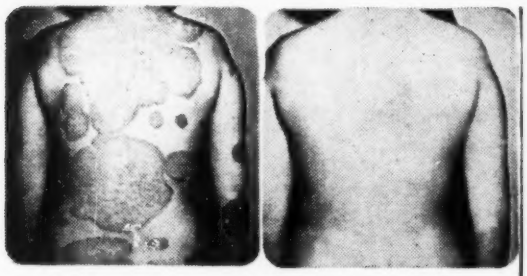
Labiton is an invigorating and palatable tonic prepared from stabilized KOLA NUTS by a special process which fully preserves the complex catechol caffeinates responsible for the specific tonic activity of the fresh nut.
Labiton is recommended in convalescence —especially after influenza. In depressive states and all forms of debility.

LABITON
REGD. TRADE MARK

In West Africa the Kola Nut is highly prized for its stimulating and restorative properties.

Literature will be gladly supplied on request.

LAB
LABORATORIES FOR APPLIED BIOLOGY LTD.
91 Amhurst Park, London, N.16. Telephone: STAmford Hill 2252/3



Adult female. Total body psoriasis of several years' duration. Alphosyl started 22/1/58. Lesions practically clear by 5/3/58.

dramatic clinical results in psoriasis

followed by medical successes throughout the United Kingdom

Though modern medicaments have brought almost dramatic relief to many forms of skin affection, Psoriasis has so far remained intractable. Its ætiology continues to baffle investigation and many forms of tried therapy have yielded only partial or temporary results.

Now ALPHOSYL — after extensive clinical trials and medical successes throughout the United Kingdom — shows promise of bringing total clearances in many cases of Psoriasis.

In ALPHOSYL, for the first time, are presented in combination two agents: allantoin and a specially refined coal tar extract. The synergistic effects of these medicaments result in rapid clearance of the condition itself and positive stimulation of the healing process.

ALPHOSYL LOTION combines these two valuable agents in one preparation and has had impressive results where other medications have failed.

The successes obtained with ALPHOSYL indicate that it deserves a trial in all cases of Psoriasis—no matter how extensive the area of infection, how long the duration of the condition, or whatever the therapy previously used. References: "Clinical Medicine" Vol. 5, No. 4, April 1958; and "Ohio State Medical Journal," Vol. 55, No. 6, June, 1959.

alphosyl LOTION

In 8 fl. oz. bottles

FOR SUB-ACUTE and chronic dermatoses

TAR + HYDROCORTISONE

combined in

TARCORTIN CREAM

have pronounced advantages over either medicament alone.

The established germicidal, stimulating and anti-pruritic properties of tar in the treatment of sub-acute and refractory skin affections, can now be considerably enhanced by its incorporation with hydrocortisone.

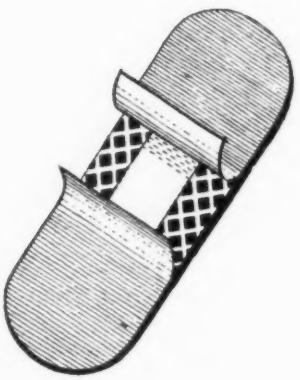
*Clinical trials have shown that the two medicaments together exert a powerful synergistic action that is far more rapid, more pronounced and complete than the action of either alone. These two valuable therapeutic agents are presented in a non-greasy, stainless hydrophilic cream, known as TARCORTIN.

TARCORTIN CREAM *is supplied in 7 grm. and 15 grm. tubes, containing 0.5% hydrocortisone in a special coal-tar extract.*

**Literature available on request.*

STAFFORD-MILLER LIMITED Manufacturing Chemists **HATFIELD · HERTS**

the only air-permeable waterproof dressing



Elastoplast Airstrip is the only dressing in the world that is waterproof *and* air-permeable. Being non-occlusive it does not cause maceration but permits the wound to dry, allowing rapid primary healing

Elastoplast Airstrip

- prevents maceration
- can be left on until the wound heals
- stretches in any direction
- antiseptic, elastic pad
- individually wrapped and sterilised

an S&N *aid to surgery*

SMITH & NEPHEW LIMITED
WELWYN GARDEN CITY, HERTFORDSHIRE

Shell achievement

One of Shell's recent achievements in Industrial Lubrication has been to reduce the risk of skin trouble for machine operators. Until the new Shell Dromus range, most modern soluble cutting oils contained phenolic compounds, which can cause skin irritation. They were used as coupling agents betweer the actual oil and the emulsifier. On high-speed machines, especially, the water evaporates and the emulsion concentrates. And that's when the trouble can start. Shell scientists found a better coupling agent, and made it work. And now, at no extra cost, management can reduce working risks for their staff. *Write for the book, 'Selecting Your Cutting Oils', to Shell-Mex House, London.*

SHELL

SHELL INDUSTRIAL OILS

A NEW EYE WASHING SERVICE

When workers' eyes are splashed with chemicals, immediate eye washing is essential to minimise the damage and pain.

This new service solves the problem of how to ensure that simple and effective eye-washing facilities are at once available at every point where the hazard exists.

The outfit is very simple to operate. There is nothing in the apparatus to go wrong. Clear, full instructions are included, and staff can quickly be trained in its use.

SANDOID
EYE-WASHING
CABINET No. A205

An important feature is the safe steady flow of eye-washing lotion; there is no forceful jet to hit and damage the eyeball.

The Cabinet illustrated is size 13¾ ins. × 13¼ ins. × 6¼ ins. and is supplied at £4 17s. 6d. Eye lotion is available in one gallon "Cubitainers" with dispensers or concentrate in 4 oz. bottles.

C1641

A205E

SEND FOR FULL DETAILS TO:

Cuxson, Gerrard and Co. Ltd.

OLDBURY — BIRMINGHAM

TELEPHONE : BROADWELL 1355

Brit. J. industr. Med., 1961, **18**, 241.

REHABILITATION: CONCEPT AND PRACTICE*

BY

J. A. L. VAUGHAN JONES

From the East and West Ridings Region of the Ministry of Labour

(RECEIVED FOR PUBLICATION OCTOBER 4, 1960)

The word "Rehabilitation" has been used in many contexts and in many different ways. In the medical field its interpretation has changed considerably in the period from the years before the first world war to that after the second and the ensuing extensive social legislation which followed the Beveridge Report. Changes in definition, although usually enlarging the scope, do not necessarily mean increased application. The multiplicity of statutory and other agencies which have a part in the method and means of returning patients to full industrial and social contact has contributed to the failure to progress with the changes in definition.

Any review of rehabilitation services in this country shows many gaps which are due primarily to a failure by the medical profession as a whole to accept the dynamic philosophy of treatment demanded by rational schemes of rehabilitation and secondarily to the failure of effective co-ordination and integration between government departments, hospitals, general practitioners, public health services, and industry.

The prevailing tendency to isolate rehabilitation in the sphere of physical medicine is criticized as an attempt to evade a responsibility which the whole practising profession must accept. The fundamental function of industry in the process and the importance of the role of the Industrial Medical Officer are emphasized. Resettlement in employment is the crux of the whole process of rehabilitation.

In choosing the title, I wondered whether to concentrate on a purely academic approach or whether to detail my experience in a particular branch of medicine relating mainly to industry. In the end I have compromised by a combination of both. First an interest in occupational health, which began as a student, was stimulated when I was thrust into the vortex of the daily casualties of a Welsh mining community. Rehabilitation was a word used in limited circles, mostly outside medicine, and understood by few, but a youthful anxiety to try to get men back to work earlier than they did in those days of 1924 was the glimmering in my own mind of our modern approach. This was no pioneer work; it had already been undertaken by Robert Jones, Agnes Hunt, Moore at Crewe, and others, but it was an instinctive reaction to the months of suffering and waiting which was the lot of many patients; waiting until the wounds healed; waiting until the claim for compensation so bitterly fought, was settled; waiting to be allowed to recover their sense of independence; waiting to be allowed to face death daily at their work. It was not the hacking coughs and the marked dyspnoea of so many which was disturbing, as much as the poor results of the traumatic surgery of the era which constituted a major challenge.

Immobilization for months in splints and later in plaster, more months spent hobbling about aimlessly trying to restore function in limbs too long rested and wasted, months spent wrangling in the Courts about differing and sometimes conflicting opinions of medical experts: months which embittered men and their families, and much of the blame could properly be stated to be the responsibility of a profession much too slow, or, to be charitable, too conservative to learn the lessons so clearly demonstrated to it by those pioneers in Britain before, during, and after the first world war.

It has been said that rehabilitation is physical medicine: how shockingly insular. I am not a physical medicine expert. Nevertheless, I have occupied a somewhat privileged position to survey the scene and watch the developments. In 1940, I became Industrial Medical Officer at a Government

*From the Mackenzie Industrial Health Lecture of the British Medical Association, delivered during the annual provincial meeting of the Association of Industrial Medical Officers, held at Southampton, September 1960.

A NEW EYE WASHING SERVICE

When workers' eyes are splashed with chemicals, immediate eye washing is essential to minimise the damage and pain.

This new service solves the problem of how to ensure that simple and effective eye-washing facilities are at once available at every point where the hazard exists.

The outfit is very simple to operate. There is nothing in the apparatus to go wrong. Clear, full instructions are included, and staff can quickly be trained in its use.

SANDOID
EYE-WASHING
CABINET No. A205

An important feature is the safe steady flow of eye-washing lotion; there is no forceful jet to hit and damage the eyeball.

The Cabinet illustrated is size 13¾ ins. × 13¼ ins. × 6¼ ins. and is supplied at £4 17s. 6d. Eye lotion is available in one gallon "Cubitainers" with dispensers or concentrate in 4 oz. bottles.

C1641

A205E

SEND FOR FULL DETAILS TO:

Cuxson, Gerrard and Co. Ltd.

OLDBURY — BIRMINGHAM

TELEPHONE: BROADWELL 1355

Brit. J. industr. Med., 1961, **18**, 241.

REHABILITATION: CONCEPT AND PRACTICE*

BY

J. A. L. VAUGHAN JONES

From the East and West Ridings Region of the Ministry of Labour

(RECEIVED FOR PUBLICATION OCTOBER 4, 1960)

The word "Rehabilitation" has been used in many contexts and in many different ways. In the medical field its interpretation has changed considerably in the period from the years before the first world war to that after the second and the ensuing extensive social legislation which followed the Beveridge Report. Changes in definition, although usually enlarging the scope, do not necessarily mean increased application. The multiplicity of statutory and other agencies which have a part in the method and means of returning patients to full industrial and social contact has contributed to the failure to progress with the changes in definition.

Any review of rehabilitation services in this country shows many gaps which are due primarily to a failure by the medical profession as a whole to accept the dynamic philosophy of treatment demanded by rational schemes of rehabilitation and secondarily to the failure of effective co-ordination and integration between government departments, hospitals, general practitioners, public health services, and industry.

The prevailing tendency to isolate rehabilitation in the sphere of physical medicine is criticized as an attempt to evade a responsibility which the whole practising profession must accept. The fundamental function of industry in the process and the importance of the role of the Industrial Medical Officer are emphasized. Resettlement in employment is the crux of the whole process of rehabilitation.

In choosing the title, I wondered whether to concentrate on a purely academic approach or whether to detail my experience in a particular branch of medicine relating mainly to industry. In the end I have compromised by a combination of both. First an interest in occupational health, which began as a student, was stimulated when I was thrust into the vortex of the daily casualties of a Welsh mining community. Rehabilitation was a word used in limited circles, mostly outside medicine, and understood by few, but a youthful anxiety to try to get men back to work earlier than they did in those days of 1924 was the glimmering in my own mind of our modern approach. This was no pioneer work; it had already been undertaken by Robert Jones, Agnes Hunt, Moore at Crewe, and others, but it was an instinctive reaction to the months of suffering and waiting which was the lot of many patients; waiting until the wounds healed; waiting until the claim for compensation so bitterly fought, was settled; waiting to be allowed to recover their sense of independence; waiting to be allowed to face death daily at their work. It was not the hacking coughs and the marked dyspnoea of so many which was disturbing, as much as the poor results of the traumatic surgery of the era which constituted a major challenge.

Immobilization for months in splints and later in plaster, more months spent hobbling about aimlessly trying to restore function in limbs too long rested and wasted, months spent wrangling in the Courts about differing and sometimes conflicting opinions of medical experts: months which embittered men and their families, and much of the blame could properly be stated to be the responsibility of a profession much too slow, or, to be charitable, too conservative to learn the lessons so clearly demonstrated to it by those pioneers in Britain before, during, and after the first world war.

It has been said that rehabilitation is physical medicine: how shockingly insular. I am not a physical medicine expert. Nevertheless, I have occupied a somewhat privileged position to survey the scene and watch the developments. In 1940, I became Industrial Medical Officer at a Government

*From the Mackenzie Industrial Health Lecture of the British Medical Association, delivered during the annual provincial meeting of the Association of Industrial Medical Officers, held at Southampton, September 1960.

Training Centre of the Ministry of Labour and National Service, training all types of people for work in the war effort, girls, women, boys, men, able-bodied and handicapped; all who could walk and breathe were potential trainees. The adaptation of down-and-outs, social outcasts, and quite severely disabled persons to economic working units was a tremendous achievement, spurred on by the urgency of the manpower need of the war, but it provided a real insight into the problem, an insight into the potential of disabled or handicapped people who had been regarded as cripples with no future. This work continued during the war years, and when peace came, was adapted to the training of ex-servicemen for peace-time needs, for men and women who had lost their opportunities through their period of war service.

History of Legislation concerning Rehabilitation

In 1941 the interim scheme of the Ministry of Labour and National Service in relation to disabled persons linked hospitals and the employment exchanges together for the first time. The Tomlinson Report was published in 1943 and greatly stimulated medical and lay interest in the problem. It envisaged a post-war scheme to deal with the casualties of war and peace, and encouraged action on the part of several government departments concerned.

In 1948 I was appointed a Regional Medical Adviser to the Ministry of Labour and National Service with the following duties:

(1) to act as Regional Medical Adviser to the Regional Controller on general medical problems arising out of resettlement work in respect of disabled persons;

(2) to make inquiries and advise, as found necessary, on special problems of that kind;

(3) to advise the Regional Controller on organization and content of medical services in vocational training and at industrial rehabilitation centres;

(4) to assist in developing the fullest co-operation within the Region between the employment service of the Ministry of Labour and the hospital and other medical services and the medical profession generally, in relation to the problem of placing disabled persons;

(5) by association with appropriate departments of universities or approved hospitals, or in other ways, to maintain a research and teaching interest in medical aspects of the placing and resettlement of disabled persons.

This appointment was made by the Ministry of Health, the holder being seconded to the Ministry of Labour. The work involved a close relationship with Regional Hospital Boards and teaching hospitals, contacts with hospital staffs in two Regions, with the local authorities of the area, and liaison with general practitioners. The University relationship had already been established by my appointment to a part-time lectureship in occupational health.

An objective view could be taken from this particular position of the attitude of medical teachers to the problems of rehabilitation, of the differing approach by two Regional Hospital Boards and the teaching hospitals together with the general reaction of the medical profession to the new demands made upon it.

Twelve years have passed and there have been many changes, but Departments work all too slowly, and at times our own profession has moved even more slowly.

The Impact of Post-war Legislation

Public reactions to the social problems of the crippled, the disabled, and the handicapped have not inherently changed, in spite of a great deal of lip-service to the concept of social welfare or to the achievement of social independence, which should be the ultimate aim. Conservatism, which can be an admirable excuse for the non-progressive in our profession, has severely limited both the potential and the ultimate successes. When distinguished members of our profession say that consultants have nothing to learn about problems of rehabilitation, that it is largely a matter of education of the general practitioner—and this is uttered in a mainly lay Departmental Committee—when other people say that the term "Rehabilitation" should be put into cold storage for 20 years, it seems doubtful whether there is any real hope that our profession will accept its social responsibility.

The concept is best approached on a chronological basis. First there is the Report of the British Medical Association Committee on Fractures which stimulated the appointment of the Delevigne Committee on the treatment of fractures, which reported in 1936-37. The B.M.A. definition of 1934 was:

"The word 'Rehabilitation' may be taken as applicable to a stage between the completion of a course of massage and exercises in the Massage Department and the point where the stresses and strains of heavy work can be undertaken. It is an active stage, directed to a complete restoration of function."

The Delevigne Committee Report resulted in the setting up of fracture clinics throughout the country, which later developed into the Emergency Medical Service, set up in 1938-39 to deal with the expected casualties of an approaching war. Under the stimulus of the imperative war-time demands for manpower, public interest was aroused by the need for the utilization of disabled or handicapped persons in the national effort, and many people realized for the first time that sub-standard individuals could contribute a full day's work if they were appropriately handled and allocated.

In 1941, Ernest Bevin, then Minister of Labour and National Service, appointed a Departmental Committee on the Rehabilitation of Disabled Persons, under the Chairmanship of George Tomlinson, who was Parliamentary Secretary to the Ministry. Their remit was:

(*a*) to make proposals for introduction, at the earliest possible date, of a scheme of rehabilitation and training for the employment of disabled persons not provided for by the interim scheme introduced by the Ministry of Labour and National Service in October 1941;

(*b*) to consider and make recommendations for the introduction, as soon as possible after the war, of a comprehensive scheme for—
(i) the rehabilitation and training of } disabled persons in all
(ii) securing satisfactory employment for } categories.

(*c*) to consider and make recommendations as to the manner in which the scheme proposed for after the war should be financed.

The Tomlinson Report has been one of the great social advances of our time; most of what has been done in this country in rehabilitation and resettlement is a direct result of its recommendations. The conclusions of the Tomlinson Report (1942) were as follows:

(1) Rehabilitation in its widest sense is a continuous process, partly in the medical sphere, partly in the social and industrial spheres. The medical side, in spite of the developments brought about under the Emergency Hospital Scheme, still falls short of what is required, and, so long as this continues to be so, the problem of rehabilitation at the post-hospital stage, is correspondingly more serious and extensive. In the meantime, concerted action is necessary to see that the existing hospital facilities are in use to the fullest extent and to the best advantage for those in need.

(2) Close co-operation in the Health and Industrial Services is necessary throughout the whole process. This is particularly important at the stage between the end of medical and the beginning of industrial rehabilitation; but it should be continued to the later stage of resettlement, so that the industrial services have the benefit of medical advice, and the medical services can acquire knowledge of the effect of disablement upon occupational capacity.

(3) On the industrial side, continuity of the service is essential. It should start in the hospital, it should continue throughout the post-hospital stages, it should not end with the first placing in employment, but should follow up until resettlement is completed. This calls for a specialist service within the Ministry of Labour and National Service.

(4) Ordinary employment is the object and is practicable for the majority of the disabled, with the goodwill and co-operation of the representative organizations of employers and workpeople, in conjunction with the Health Services and the responsible Government Departments.

(5) A minority of the disabled require employment under sheltered conditions, and such employment should be provided through production for Government and other public services.

These conclusions led the Committee to their final recommendation which was for the establishment of machinery to augment the work of the Departments responsible for the many different aspects of the whole scheme. These Departments were: Ministry of Labour and National Service, Ministry of Health, The Board of Education, together with the Scottish Departments of Health and Education, Ministry of Pensions, The Home Office, and Ministry of Fuel and Power.

It was suggested that an inter-Departmental Committee should be appointed forthwith to ensure that adequate preparations were made for the introduction of the scheme immediately after the war. The recommendations of the Committee dealt with eligibility, medical rehabilitation, post-hospital rehabilitation, resettlement, employment under sheltered conditions, employment of the blind and the deaf, employment on own account, placing and follow-up work, survey of occupations, and finance.

The following extract from the Tomlinson Report is relevant to the concept:

" The successful rehabilitation of a person disabled by injury or sickness is not solely a medical problem. Rehabilitation in its strictly medical sense means a process of preventing or restoring the loss of muscle tone, restoring the full function of the limbs, and maintaining the patient's general health and strength. The process should begin as soon as possible after injury or operation, and, in the case of acute or prolonged illness, as soon as the patient's condition permits, and it should continue not only throughout the period of hospital treatment, but also during the subsequent stage of convalescence, whether that takes place in hospital, or is provided in a separate centre. Continuity of treatment is essential to achieve the aim of restoring a patient's mental and physical capacity at the earliest possible date, and to the fullest possible extent."

In 1943 the Reports were published of the Scottish Experiments in Social Medicine at Gleneagles (later at Bridge of Earn) and that on the development of the Miners' Welfare Rehabilitation Centres.

In 1944 the Disabled Persons (Employment) Act was passed. It was the first positive step taken as a result of the Tomlinson Report, and much of the subsequent work of the Ministry of Labour and National Service flowed from it.

In 1946 a British Medical Association Committee reported on Rehabilitation and defined the problem as follows:

" Rehabilitation is the fullest possible restoration to normal life and working efficiency of a person incapacitated by disease or injury. This is primarily a medical problem, but one in which industry and the social services must play an essential part. Treatment must be directed not only to obtaining full physical and mental recovery, but also to restoring a patient's enjoyment of his former cultural and social activity and his resettlement in work best suited to his capacity."

In 1949 the Ministry of Health, in a circular sent to all Hospital Boards, Boards of Governors, and Hospital Management Committees, urged the medical profession to accept an additional social responsibility in relation to their patients. The circular stated that treatment should not end with the departure of a patient from hospital, or with the getting up out of bed after severe illness, but should be continued into the stage of resettlement in appropriate work. Further, that treatment was the responsibility of the hospitals and the medical profession, but that placing in work was quite clearly the responsibility of the Ministry of Labour and National Service through its placement and Disabled Resettlement Officer Services: an excellent concept, but its implementation has not been achieved. At the same time it was urged that all larger hospitals should appoint one or two senior members of the staff to supervise rehabilitation services within the hospital or hospital group. It is unfortunate that the medical profession found in this apparent contradiction between the acceptance of individual responsibility and the delegation of responsibility, an excuse for the lack of progress which should have been made. The result was that the profession accepted individual responsibility in theory, but was opposed to the appointment of colleagues with additional responsibility. That situation is largely maintained today.

In 1954, the B.M.A. second Committee on Rehabilitation, in giving evidence to the Piercy Committee, produced this definition:

" The rehabilitation of a patient is an indivisible process beginning with the onset of sickness or injury, and continuing throughout treatment until final resettlement in the most suitable work and living conditions is achieved. Its aim is to enable him to resume his place as a responsible citizen, and to contribute in the fullest possible manner to the physical, mental and social welfare of the community. The whole man must be rehabilitated, the patient being dealt with as an individual with a distinctive constitutional make-up, with his own emotional life and moral values and with a particular upbringing and social and economic background. In this sense, every aspect of medical care is a part of rehabilitation."

The Report goes on to say:

" It is this comprehensive conception of rehabilitation which places upon the medical profession special responsibility, for only a person who is medically trained can advise the patient with a full understanding of his disability. Similarly, the complete confidence of the patient will be gained only if he can feel that he is being guided by someone who understands all aspects of his problem. There must, therefore, be continuous medical supervision of the patient throughout the process of rehabilitation."

In 1955, the Report of the expert Committee on Medical Rehabilitation of the World Health Organization made a statement of general aims and principles, which were virtually in accord with the definitions of the British Medical Association, and finally there was the Report in 1956, of the United Nations Working Party on the Rehabilitation of the Physically Handicapped. The following is an extract from this Report:

" The time has long passed when a handicapped child or a disabled adult should be regarded as a subject for commercial exploitation, and trained for the occupation as a professional beggar, or even to be considered as a mere object of charity. Modern methods of medical and sociological science have opened a new horizon of promise for such individuals, but if this promise is to be fulfilled and the handicapped person is to have his full chance in life, there must first be a new evaluation of physical disability, based on the following six theses:

(1) That the handicapped person is an individual with full human responsibilities which he shares in common with the able-bodied, and that he or she is entitled to receive from his or her country every possible measure of protection, assistance and opportunity for rehabilitation.

(2) That, by the very nature of his physical handicap, he is exposed to the danger of emotional and psychological disturbance resulting from a deep sense of deprivation and frustration, and that he therefore has special claims on society for sympathy and constructive help.

(3) That he is capable of developing his residual resources to an unexpected degree, if given the right opportunity for so doing, and of becoming in most instances an economic asset to the country instead of being a burden on himself, his family and the State.

(4) That handicapped persons have a responsibility to the community to contribute their services to the economic welfare of the nation, in a way that becomes possible after rehabilitation and training.

(5) That the chief longing of the physically handicapped is to achieve independence within a normal community instead of spending the rest of his life in a segregated institution, or within an environment of disability.

(6) That rehabilitation of the physically handicapped can only be successfully accomplished by a combination of medical, educational, social and vocational services, working together as a team."

The changing attitudes expressed in these definitions indicate the developments that have taken place in the approach to rehabilitation. Nevertheless, unless some really positive action results from the dissemination of these formative ideas, definitions tend to remain mere skeletons. To appreciate all the agencies involved in the problem of the transfer of man from bed to work provides what is probably the real reason for a comparative failure of the service in this country. The word " comparative " is used lightly, because we are still ahead of many countries; we could have been ahead of all in practice as well as theory if a little more positive action had been taken.

The numerous bodies concerned with rehabilitation include the statutory authorities, the Ministries of Education, Health, Labour, Pensions and National Insurance, the National Assistance Board, the Hospital Boards of the National Health Service, the General Practitioner Service, the Public Health Service, and the Local Authority Health and Welfare Service. A host of voluntary bodies contribute their parts to the solution of the problem; also universities, industry, trade unions, employers' organizations and, last, but not least, industrial medical officers.

The Present Position

It is the lack of integration of all these services which has been the main delaying factor in the proper development of the scheme. If the problems of the different services are envisaged and particularly those of our own profession, it is realized first of all that there is an absence of modern thought in the teaching stage when the medical student is at his most impressionable age. The problems of the National Health Service have almost engulfed the medical profession, which consequently has failed to appreciate the vital contribution which a dynamic philosophy of treatment can achieve. It has shown a lack of vision or even a desire to eschew anything which appears new (apart from drugs) and an almost fanatical belief in the methods of the past. It may be that the increasing fragmentation of medicine, particularly of the specialties, may have played a part in this. The welter of post-war social legislation has almost submerged all except the cynical, the embittered, and the angry, but positive constructive thought or action rarely emerges from these groups.

In spite of the brilliant achievements of a few enlightened doctors who had the courage of their convictions, too little has been achieved in the sphere of rehabilitation. Platitudes, professional jealousies, empire building on the part of lesser known specialties, apathy, and plain ignorance are charges which have to be met. One expert has divided the medical profession into five groups on their attitudes to rehabilitation: tolerant 30%, passive 50%, interested 15%, enthusiastic 3%, expert 2%.

The past 12 years have been spent trying to educate, to encourage, to cajole, even to force, colleagues to appreciate that medicine now includes social responsibilities towards patients. These responsibilities must be accepted as a fundamental part of the modern concept of treatment in that the work is not completed until the patient is back at work, properly settled in appropriate employment. This new concept of treatment also demands that doctors are part of a team which involves many other agencies. Doctors are essentially individualists, but occupational health shows that success can be achieved only as members of a team; this is equally true in the concept of rehabilitation.

There is one bright light on the horizon, and that is the avidity with which final year medical students accept the philosophy of rehabilitation and ask for more in the few lectures which are allowed in the curriculum. The tragedy is that rehabilitation should not be taught as a specialist subject, but as an inherent part of all clinical teaching; that will be too much to expect of this decade. Nevertheless, junior resident medical staff are more ready to accept this wider concept than their seniors, and one Regional Hospital Board at least, has instituted courses in rehabilitation for junior resident hospital medical officers during their first two years. This sensible action should be copied nationally.

Many general practitioners are more knowledgeable about this problem than is generally realized.

Industrial medical officers play an important part in the resettlement of the individual returning to industry; the industrial medical officer often has the extremely difficult task of relating an employee's altered capacity to the jobs which are available. The detailed results of the follow-up of these cases once they are back in industry can assist the other services engaged in rehabilitation to a very considerable extent; in addition the information available from the industrial medical officer has not been fully used.

To turn now to the statutory authorities. The Ministry of Labour, under the impetus of the Disabled Persons (Employment) Act, has probably made a greater contribution to the subject of rehabilitation than any other Department by the provision of Disabled Resettlement Officers at every local Employment Office, a corps whose training has been steadily increased and improved since 1944; by its Industrial Rehabilitation Units which carry out the work of reconditioning people returning to their own or other work or assessing cases requiring a change of work or training; by its Medical Interviewing Committees which give medical advice to

assist the D.R.O. in the placing of disabled persons; by its Government Training Centres, which train both disabled and able-bodied for new vocations, and its Remploy Factories for the more seriously disabled; and by its Disablement Advisory Committees, dealing with local problems.

In recent years the Ministry of Health, particularly since the publication of the Piercy Committee Report, has advised Regional Hospital Boards, Boards of Governors, and Local Authorities to tackle the problem of rehabilitation, the problem of handicapped persons of all ages. The Ministry has issued documents to the Hospital Service, the Local Authorities, and to general practitioners on the general problems of rehabilitation, and has encouraged Boards and Hospitals particularly concerned, not only to develop, but to make the best use of existing facilities and to provide facilities for a system of planned convalescence. The streamlining of these rehabilitation services run under the aegis of the Ministry of Health could save the National Health Service millions of pounds and would reduce the demand for more hospital beds which is often made. The increased use of this new concept of treatment means a shorter hospital stay and a quicker return to work. These are basic factors in the national economy.

The Ministry of Health in a Circular to Regional Hospital Boards and Boards of Governors stated:

" Rehabilitation is not to be regarded as the application of special techniques, and still less as a separate medical or other specialty, but above all, as a constituent part of the thought and action of all those who are concerned with treating patients and the restoration of disabled persons to their utmost capacity."

In spite of this, the Ministry of Health in its circulars has tended to concentrate on a thesis of physical medicine as expressed by physiotherapy, remedial gymnastic, and occupational therapy services, rather than to demand from the medical profession the individual and collective response which is its right. It is important not to build a rehabilitation service entirely on a basis of physical medicine. Of the groups which comprise the profession, the orthopaedic, traumatic, and thoracic surgeons, the chest physicians, the psychiatrists, and the physical medicine group have shown a greater willingness to accept the additional responsibilities of treatment than others, but it is emphasized that rehabilitation is one of every doctor's fundamental responsibilities.

Both Departments have encouraged the use of resettlement clinics or case conferences at every major hospital, *i.e.* clinics in which consultant, almoner, disabled resettlement officer, and doctor with knowledge of local industry can pool their knowledge to the benefit of the patient, but the response has been completely inadequate.

The Piercy Committee, a mainly inter-Departmental Committee, was set up by the Ministers of Labour and Health and the Secretary of State for Scotland in 1953 with the following terms of reference:

To review in all its aspects the existing provisions for the rehabilitation, training, and resettlement of disabled persons, full regard being had for the utmost economy in the Government's contribution, and to make recommendations.

The need for economy will be noted. This Committee arose from the Government's wish to ascertain what had happened as a result of the numerous legislative measures passed since the Tomlinson Report of 1943. The Committee finally reported in September 1956, after holding 54 meetings and taking voluminous evidence. In the interpretation of its terms of reference there was a significant change in the definition of rehabilitation. Paragraph 5 of the Piercy Committee's Report reads:

" The term ' Rehabilitation ' in its widest sense signifies the whole of the process of restoring a disabled person to a condition in which he is able, as early as possible, to resume a normal life. With this interpretation, it would cover also training and resettlement in employment. As the terms of reference specifically mention rehabilitation, training and resettlement, the Committee decided that its use of the term ' Rehabilitation ' should be confined to medical and surgical treatment designed to restore physical and mental functions, and to the process of reconditioning, designed to restore the capacity for taking up employment or vocational training. Whilst the Committee was concerned primarily with vocational training for the disabled, it decided to take account also of other forms of training of the disabled more strictly educational in content. It also decided that the term ' Resettlement ' should be used to cover both the placing of disabled persons in employment (including any follow-up action to ascertain the effectiveness of the placing) and action taken to re-settle other disabled persons—such as housewives—who are not in the industrial field."

The separation of the terms " Rehabilitation ", " Training ", and " Resettlement " may indicate a desire to preserve Departmental tidiness, but this fragmentation tends to promote gaps when the whole process should be continuous.

The Piercy Report found omissions and deficiencies in almost every sphere and in every agency involved; deficiencies not only of personnel but also in the appreciation of the situation and its problems. Referring to administrative arrangements, the Report says (paragraph 317):

" To secure the most efficient operation of these services (from the standpoint of the individual) at the minimum

cost in manpower and money, sensitive contact and willing co-operation are necessary between the various agencies and Departments concerned. This is needed at the centre, at the local level and among the regional authorities. The Committee believes that some improvement in existing conditions at all levels may be desirable, and is attainable."

This is a most masterly understatement.

The Committee finally made 46 recommendations, covering Hospital Services, Resettlement Clinics, Regional Hospital Boards, Rehabilitation Committees, Industrial Rehabilitation, Comprehensive Rehabilitation and Assessment Centres, Rehabilitation Services provided by employers, Welfare Services, appliances and other aids for the disabled; Vocational Training, Disabled Persons' Register, Placing of the Disabled in Employment, Sheltered Employment including Remploy, Home Workers' Schemes and provisions for the young and other special categories of the disabled.

The Report was clearly a challenge to all participating authorities, but subsequent action was considerably delayed. It was not until 1958 that the Ministry of Health issued Circular H.M. (58) 57 on *Rehabilitation in the Hospital Service and its Relation to other Services*, and Circular 16/58 to Local Authorities on *Services for Handicapped Persons*. The Circular to general practitioners on *Rehabilitation of the Sick and Injured* appeared in July 1959, but was not circulated until further information as to available local facilities for rehabilitation was included as an appendix.

Several agencies, including Hospital Boards and Local Authorities, took positive action before the issue of these circulars. Indeed one Regional Hospital Board had taken in 1949-50 the action proposed by the second of the Piercy recommendations. The Regional Rehabilitation plan, evolved at that time by the co-operation of the Leeds Regional Hospital Board and the East and West Ridings Region of the Ministry of Labour, had reposed in the files of the appropriate Ministries since that time although the Chief Medical Officer of the Ministry of Health had expressed the view that this plan should be the pattern for all Regional Rehabilitation Services. It is interesting to relate that one Department denied that it had a copy of the plan.

Other Regional Hospital Boards are now engaged in an attempt to implement the recommendations of the Piercy Committee, and serious efforts are being made to improve the content of services, and to co-ordinate the various services. These efforts do not necessarily take the same pattern; for example, two adjoining Regional Hospital Boards have entirely different concepts. One believes that all rehabilitation services should be based on the establishment of physical medicine consultants and centres at strategic points in the Region; the other Board feels that one of its basic functions is to implant the ideology and philosophy of rehabilitation into all its consultants and hospitals. The results will be watched with considerable interest.

The Future

Making the assumptions:

(1) that hospital and general practitioner services can be improved to the extent that the responsibilities of rehabilitation are fully appreciated;
(2) that Local Authorities accept in full their responsibilities under the various enactments;
(3) that the various Ministries co-ordinate the work more efficiently and provide the necessary stimuli from time to time,

what remains to be done, and what should be done to anticipate the demand on other resources?

In the first place, is there the necessary link with industry to obtain resettlement? The link is probably there, but will not be efficient until there is a comprehensive Occupational Health Service in this country. Some large firms have achieved much in this sphere on behalf of their own workers, but at present their achievements cannot for numerous reasons be copied by the smaller undertakings.

There is a real need for the appreciation by all those engaged in the service of rehabilitation (both medical and lay) that entry into industry (and re-entry into industry) is no longer a slap-happy event, even although that term may still be applied to a number of school leavers.

The relationship of the functional analysis of the worker to job analysis is an essential in the resettlement of the handicapped person. The techniques concerned should not be reserved for a small number of doctors who understand how to complete an official Ministry of Labour D.P. form and the disabled resettlement officers who frequently have to try to interpret the inadequacies of the medical profession in the completion of these forms.

It is not sufficient for the almoner at the behest of the disabled resettlement officers to chase the consultant to complete one of these forms, which may then be passed on to the senior registrar, the senior house officer, and finally the junior house officer, who cannot pass it on to anyone else. The problem of equating a man's residual capacity to a particular job is often difficult, but until and unless the medical profession learns more of the pattern and reality of industry, schemes of rehabilitation will never attain the success they merit.

It may be argued that the simplest method for dealing with the problem would be to leave it to be

settled by a few trained specialists. In my view this would be entirely wrong, because the philosophy of rehabilitation and the purpose behind it must reach and be accepted by all branches of the medical profession.

The work of the industrial medical officer in this sphere would assume reasonable proportions if a much clearer link existed between the agencies of the National Health Service and industry than at present. The growing tendency of the National Health Service and the Industrial Health Service to develop separate entities with little or no co-ordination is unsatisfactory.

Rehabilitation services will expand in this country although rather more slowly than some would like; industrial medicine and occupational health will grow, perhaps more quickly, under the stimulus of knowledgeable and visionary Ministers of Labour and Health, but they will never be complementary to each other until doctors in the consultant and general practice services fully comprehend what employment or work actually means; what industrial processes demand, and, not least, that work therapy in the actual course of employment can contribute a great deal more to the national economy and the worker's personal happiness than hours spent weekly doing elementary exercises in a hospital physiotherapy department. Nothing is more tiresome than to see the constant reference of people to hospital for exercises which can be done, and indeed are done, in the active performance of their ordinary work, and this wasted time must stop.

The responsibilities of industry are great, because it must be impressed on the curative services of this country that the end result of all their efforts is work, and work as quickly as possible; the social aspect will follow.

Employers and workers alike must not put barriers in the way of resettlement of handicapped or disabled persons. The limitation of jobs, the demarcation of jobs, the petty differences, the lack of liaison between trade unions in relation to a man's change of job—all these must be overcome in the knowledge that most disabled persons can contribute a full day's work and in the need to return the worker to proper employment and his social environment.

The industrial medical officer has a special responsibility for initiating schemes of liaison within his own establishment. It is part of his normal function, but it is sometimes neglected. Also he has a most important role in attempting to educate his professional colleagues about the actual performance of processes which are merely names to the majority of doctors, and in the elimination of the term " light work ".

The Piercy Committee reported that:

" Employers can—and in many cases do—take steps to modify the conditions or tempo of employment so as to enable the disabled person on return to employment after illness and injury to accustom himself more gradually to industrial conditions."

Yet there are instances where an almoner has contacted an employer in relation to a person who has had a severe illness or injury and has mentioned " light work ". Such a patient usually gets light work, and, in the absence of an industrial medical officer, often remains at the menial task supplied by the use of this archaic term. This fault is not confined to almoners.

The statement made at the seventh World Congress of the International Society for the Care of Cripples by David A. Morse—Director General of the International Labour Organization—is of the greatest importance:

" We all recognize that rehabilitation is not an end in itself, but only a means to an end. Its purpose is to change the disabled person from a state of dependence to one of independence, from disability and helplessness back to ability and usefulness at work. Resettlement in employment then is the crux of the whole process of rehabilitation."

Historians tell us that one of the greatest achievements of mediaeval Christendom was its success in making work socially satisfying. Nowadays, we seem to have lost the impetus of a faith. By the acceptance of a dynamic philosophy in relation to rehabilitation, we can really make work socially satisfying.

Bibliography

Interim Report of the Inter-departmental Committee on the Rehabilitation of Persons injured by Accidents. (1937). Home Office, Ministry of Health, and Scottish Office.

Final Report of the Inter-departmental Committee on the Rehabilitation of Persons injured by Accidents. (1939). Home Office, Ministry of Health, and Scottish Office.

Memorandum of Evidence for submission to Royal Commission on Workmen's Compensation. (1940). British Medical Association.

Report of Committee on Industrial Health in Factories. (1941). British Medical Association.

Health and Industrial Efficiency. Scottish Experiments in Social Medicine. (1943). Department of Health for Scotland.

The Organization of a Hospital Rehabilitation Department (Emergency Medical Services: Memorandum No. 6). (1943). Ministry of Health.

Rehabilitation Treatment for Injured Coal Miners. (1943). Miners' Welfare Commission.

Report of Inter-departmental Committee on the Rehabilitation and Resettlement of Disabled Persons. Cmd. 6415. (1943). Ministry of Labour and National Service.

Disabled Persons (Employment) Act. (1944). Ministry of Labour and National Service.

Second Interim Report on Social and Preventive Medicine. (1945). Royal College of Physicians, England.

Rehabilitation: The Report of the Medical Advisory Committee (Scotland). (1946). Department of Health for Scotland.

The Placement of the Disabled. (1946). *Brit. med. J.*, **1**, 803.

Rehabilitation. (1946). *Ibid.*, **1**, 990.

Modern Methods of Rehabilitation of the Adult Disabled: Report of a Group-training Course held in Sweden, Finland and Denmark. (1952). United Nations with W.H.O. and I.L.O.

The Rehabilitation and Resettlement of Disabled Persons. (1954). British Medical Association.

The Attitude of the Physician Towards Rehabilitation, chapter by Shands, A. R., in *Changing Attitudes Towards the Disabled*, pp. 26-29. (1954). Proceedings of the Sixth World Congress of International Society for the Welfare of Cripples, The Hague.

Report of the Committee of Inquiry on the Rehabilitation, Training and Resettlement of Disabled Persons. Cmd. 9883. (1956). Ministry of Labour and National Service.

Ways Back to Working Life, chapter by Morse, D. A., in *Planning for Victory over Disablement*, pp. 262-270. (1957). Proceedings of the Seventh World Congress of International Society for the Welfare of Cripples, London.

Expert Committee on Medical Rehabilitation, First Report. (1958). *Wld Hlth Org. Techn. Rep. Ser.* No. 158.

National Health Service. Rehabilitation in the Hospital Service and its Relation to Other Services. H.M. (58) 57. (1958). Ministry of Health.

Rehabilitation of the Sick and Injured (*with Central Health Services Council*). (1959). Ministry of Health.

Convalescent Treatment: Report of a Working Party. H.M. 59/53. (1959). Ministry of Health. H.M.S.O.

Unpublished Reports

Responsibility of the National Health Service in Relation to Disability. R.H.B. 49/36, H.M.C. 49/26, B.G. 49/29. Ministry of Health.

Welfare Services for Handicapped Persons other than the Blind and Partially Sighted. 32/51. Ministry of Health.

Services for Handicapped Persons. 16/58. Ministry of Health.

Training and Resettlement of Disabled Persons. PL/1941. Ministry of Labour and National Service.

Fracture Treatment and Rehabilitation. PL.113/1942. Ministry of Labour and National Service.

Brit. J. industr. Med., 1961, **18**, 250.

AIR POLLUTION IN ROAD TUNNELS

BY

R. E. WALLER, B. T. COMMINS, and P. J. LAWTHER

From the Medical Research Council Air Pollution Research Unit, St. Bartholomew's Hospital, London

(RECEIVED FOR PUBLICATION MAY 3, 1961)

As a part of a study of pollution of the air by motor vehicles, measurements have been made in two London road tunnels during periods of high traffic density. The concentrations of smoke and polycyclic hydrocarbons found there are much higher than the average values in Central London, but they are of the same order of magnitude as those occurring during temperature inversions on winter evenings when smoke from coal fires accumulates at a low level.

An attempt has been made to relate the concentration of each pollutant to the type and amount of traffic. Both diesel and petrol vehicles make some contribution to the amounts of smoke and polycyclic hydrocarbons found in the tunnels, but in the case of smoke, fluoranthene, 1 : 2-benzpyrene, pyrene, and 3 : 4-benzpyrene, the concentrations appear to be more closely related to the density of diesel traffic than to that of petrol traffic. The concentrations of lead and carbon monoxide have also been determined, and these are very closely related to the density of petrol traffic. During the morning and evening rush hours the mean concentration of carbon monoxide was just over 100 p.p.m. and peak values up to 500 p.p.m. were recorded at times. Oxides of nitrogen were determined in some of the experiments and there was always much more nitric oxide than nitrogen dioxide. Eye irritation was experienced but its cause was not investigated.

The concentration of pollution in the tunnels does not appear to be high enough to create any special hazards for short-term exposures. The amosphere at peak periods may become very dirty and unpleasant and the concentration of carbon monoxide would be sufficient to produce some effect over a period of several hours' continuous exposure. The total emission of pollution from road vehicles must still be small in comparison with that from coal fires, but the effect of traffic on the concentration of smoke, polycyclic hydrocarbons, carbon monoxide, and lead in the air of city streets deserves continued study.

Although the combustion of coal is still responsible for most of the air pollution in British towns, the contribution made by motor vehicles deserves careful attention. In an earlier paper (Commins, Waller, and Lawther, 1957) the concentrations of smoke and polycyclic hydrocarbons in two London diesel bus garages were compared with those in the outside air. In each case there was much more black smoke inside the garage than outside, but the differences in 3:4-benzpyrene concentration were small. The buses were in excellent mechanical condition and they were being run under minimal load. The results of this investigation were relevant to the health of the workers in the garages, but they were of strictly limited value in any study of the possible effects on the general population of pollution from vehicles.

The highest concentrations of pollution from motor vehicles occur in tunnels, and to study the emissions from mixed traffic, measurements were made in two London road tunnels under the Thames. Our earlier experiments had shown that the smoke from buses contains much lower proportions of polycyclic hydrocarbons than domestic coal smoke, and to avoid any interference from the latter the experiments were confined to warm days during the summer months.

Sampling Sites

The Blackwall and Rotherhithe tunnels carry all types of traffic, including regular bus services, private coaches, lorries, cars, and motor cycles. They are open also to pedestrians and cyclists, but are little used by them. Cleaning and maintenance staff spend limited periods in the tunnels and any major work is done when there is little traffic. There is no routine traffic control in either tunnel.

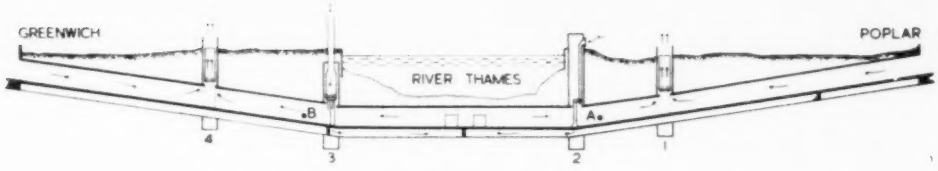


FIG. 1.—Section through Blackwall Tunnel, showing ventilation system.

At the time of these studies the Blackwall Tunnel was normally busier than the Rotherhithe and, because higher pollution was expected, most of our measurements were made there. There are corners and gradients to be negotiated in each tunnel and driving conditions are similar to those in highly congested streets. The composition of the traffic varies with the day of the week and with the time of day, and we took advantage of this fact to try to assess the separate contributions of petrol and diesel traffic to the pollution of the tunnel atmosphere. At all times there are fewer diesel than petrol vehicles, the proportion varying between about 2% on Sunday evening to over 30% at noon on weekdays.

The Blackwall Tunnel (Fig. 1) is 1,490 yards long and it connects the busy dock area around Poplar with Greenwich and the roads to the Kent coast. When opened in 1897 it was ventilated by four shafts, each 46 ft. in diameter, but fans had to be installed in them at intervals between 1922 and 1940 as the volume of motor traffic increased. The fans can be operated separately, but usually all are in use throughout the day, some are switched off in the evening, and all are off at night. The main requirement is to keep the concentration of carbon monoxide within reasonable limits, but this is sometimes difficult during prolonged hold-ups and drivers are warned to switch off their engines in these conditions. Smoke haze is usually visible in the tunnel and the atmosphere is often irritant to the eyes. Fresh air is ducted to the centre of the tunnel and also enters at the ends. The extractor fans in shafts 1 and 4 draw air from each direction, but in practice the section between the centre of the tunnel and shaft 4 is poorly ventilated. The maximum emission of pollution occurs near the bends at the foot of each shaft, where large vehicles often have to halt and then pull away up the inclines. Strong winds blowing into the ends of the tunnel can alter the pattern of ventilation, but pollution appeared to be highest between shafts 3 and 4 or between 1 and 2. The sampling points were sited in one of the sections having high smoke pollution, their exact position being dictated by considerations of safety and accessibility. In the first series of experiments (1958) the samplers were placed on a shelf 7 ft. above pavement level at a point A (Fig. 1), and in the second series (1959) they were placed on a similar shelf at point B.

The Rotherhithe Tunnel is 1,620 yards long and it connects Stepney and Bermondsey. It was opened in 1908 and fans were installed between 1924 and 1932. The general layout is similar to that of the Blackwall Tunnel, but the bends do not seriously interfere with the flow of traffic. During the period of this study the Blackwall Tunnel was closed throughout some week-ends whilst alterations were being made to the approach road, and the Rotherhithe Tunnel then carried much extra traffic. On one of these occasions samples were taken there at a point between two of the shafts, where pollution was high, using a shelf mounted 7 ft. above pavement level as in the Blackwall Tunnel.

The pollution problems encountered in these tunnels during the period 1928 to 1931 when the ventilation systems were being redesigned have been discussed by Regan (1932).

Traffic Counts

Throughout each of the experiments the flow of traffic was determined by observers standing inside or just outside the tunnel. The counts were divided into consecutive 10-minute periods, figures for each direction being recorded separately, but for most purposes the results were later expressed as vehicles per hour in either direction. Initially the vehicles were classified as "private" or "commercial" to give some indication of the proportion of petrol and diesel vehicles. Virtually all private vehicles have petrol engines and more detailed counts showed that approximately 50% of the commercial vehicles using the tunnel had diesel engines. After studying the types of vehicles in common use and drawing up a list of features to aid rapid recognition of diesel vehicles it became possible to count "petrol" or "diesel" directly and all the figures in Tables 1 to 4 have been expressed in this form.

Experience soon showed that the flow of traffic did not provide an adequate index of the emission of pollution in the tunnel. This is likely to vary with the number of vehicles in the tunnel at a given instant

and there may be a high traffic density even if the flow in vehicles per hour is low or nil. The most relevant parameter appeared to be the number of vehicles within the ventilation section in which we were working. In some experiments this was measured directly by observers stationed at each end of the section, who were in contact by telephone. When an easily recognizable vehicle passed one observer, he sent a message to the second observer who then counted and classified all vehicles passing him until the "marker" vehicle reached him. To avoid possible bias due to the selection of diesel vehicles as markers this technique was abandoned for purposes of defining the composition of the traffic. In general, it was simpler to time vehicles over the section. When necessary this was done by the two observers connected by telephone but often it was possible for one observer to walk alongside the vehicles or to stand half-way watching vehicles entering and leaving the section. The timings were made frequently and each direction was recorded separately in the first instance. From these figures mean times were calculated for each sampling period. The ventilation section considered here was that between shafts 1 and 2 (146 yards) when sampling at point A and between shafts 3 and 4 (201 yards) when at B. An estimate of the traffic density during each period was then obtained by multiplying the flow in vehicles per hour by the mean time in fractions of an hour taken to traverse the section. Results of the later experiments showed that this provided a useful statistic, although it did not take into account any variations in the emission of pollution with running conditions. The speeds of the vehicles were calculated from the times and the mean figures are quoted in Tables 1 to 4.

Procedure

During the summer of 1958 four visits were made to the Blackwall Tunnel and one to the Rotherhithe Tunnel. Congestion appeared to be greatest during the morning rush hour and samples were taken at this time on July 3 and August 20. An evening rush hour (August 1) was also studied, for there was at that time a greater proportion of petrol-engined traffic. The proportion may have been higher than on an average evening since it was the Friday before a Bank Holiday week-end. In an attempt to study almost "pure" petrol traffic further samples were taken during the late evening on Bank Holiday, August 4, when thousands of cars were returning from the coast. The traffic was fairly fast on this occasion, for it was mainly in one direction and there were few large vehicles to cause obstruction at the bends. There were some diesel buses and coaches but few lorries. All the samples up to this time were collected at point A (Fig. 1). A similar sample was taken in the Rotherhithe Tunnel on September 14 when the Blackwall Tunnel was closed and there were again thousands of cars returning from the coast on a warm Sunday evening.

An analysis of the results of these preliminary experiments showed that there was a general relationship between the concentration of each pollutant and the density of traffic, but the large number of variable factors precluded any attempt to discrimininate between the effects of petrol and diesel traffic. Big differences in the type or amount of traffic were attended by big differences in speed, and variations in wind speed and direction from one day to another were sufficient to cause differences in ventilation rate at our sampling point.

Further experiments were deferred until the summer of 1959, but in the meantime additional traffic counts were made and the pattern of pollution along the tunnel was checked visually on a number of occasions. High pollution was encountered more often at the southern end of the tunnel than at the northern, and the sampling shelf was moved to point B (Fig. 1).

To minimize the effects of variations in ventilation rate a series of experiments was planned for a single day when there was a steady wind. It was found that on a normal week-day the number of diesel vehicles using the tunnel reached a maximum towards midday and the number of petrol vehicles was highest around 6 p.m. Arrangements were therefore made to sample continuously from 10 a.m. until 7 p.m. so as to include these two peaks. To avoid interference from domestic smoke the experiment was scheduled for a very warm day when few fires would be in use. The weather became fine and warm in early May and the experiment was carried out on Thursday May 14 when the outside temperature reached 70°F. and the wind remained steady at about 5 m.p.h. from a north-easterly direction. An intensive study of pollution was made on this occasion and a team of four observers timed and counted traffic continuously. There would have been some advantage in continuing for a complete 24-hour period, but the experiments proved to be very arduous under the conditions in the tunnel, and with the staff available it was not practicable to continue any longer.

A final experiment was planned to cover an overnight period which we had not previously studied. This was started during the evening of Sunday July 5, one of the hottest days of the year, and lasted until 10 a.m. on Monday. We were thus able to examine the pollution caused by a period of very heavy traffic, consisting mainly of private cars returning from the coast, followed by a quiet period during

TABLE I

RESULTS OF TUNNEL EXPERIMENTS, SUMMER 1958

	Blackwall Tunnel								Rotherhithe Tunnel	Merton Garage
	July 3, 1958		August 1, 1958			August 4, 1958	August 20, 1958		September 14, 1958†	June 21, 1956
Time	08·45-09·45	09·50-10·50	16·00-17·15	17·33-18·33	18·38-19·38	19·30-22·00	08·30-09·36	09·38-10·32	21·00-22·42	23·00-01·00
Traffic flow*: Petrol	919	631	1,037	1,228	1,054	1,552	1,136	896	1,439	—
Diesel	272	233	228	210	140	55	335	312	125	—
Speed (m.p.h.)	—	—	—	1·7	2·9	4·1	2·0	2·8	16·2	—
Traffic density†:										
Petrol	—	—	—	60	30	31	47	26	20	—
Diesel	—	—	—	10	4	1	14	9	2	—
Carbon monoxide (p.p.m.)	295	—	—	200	—	138	—	70	170	—
Smoke (mg./100 m.³)	235	174	133	162	116	93	157	179	191	144
Hydrocarbons, μg./100m.³ air:										
Fluoranthene	73	66	29	38	21	42	35	27	54	10
1 : 2-benzpyrene	19	16	9	16	10	11	11	12	17	2
Pyrene	119	100	43	62	30	60	52	39	81	17
Coronene	35	33	17	34	25	22	22	21	30	—
Anthanthrene	16	17	6	17	12	13	11	9	18	—
1 : 12-benzperylene	53	53	19	57	42	36	40	37	55	2
3 : 4-benzpyrene	29	26	14	24	12	18	17	16	27	3
Hydrocarbons, μg./g. smoke:										
Fluoranthene	308	381	216	233	184	434	225	151	283	69
1 : 2-benzpyrene	80	95	69	98	87	117	71	68	89	17
Pyrene	505	578	324	387	257	628	328	216	425	120
Coronene	150	191	125	213	215	228	142	116	155	—
Anthanthrene	67	97	44	102	106	132	70	52	94	—
1 : 12-benzperylene	225	307	140	352	361	377	352	208	295	13
3 : 4-benzpyrene	123	151	109	146	101	186	111	91	141	18

*Vehicles per hour in both directions.
†Estimate of number of vehicles in relevant ventilation section (see text).

the night after which there was the morning rush hour. The flow of cars continued well into the night, but our hopes of sampling "pure" petrol engine pollution at that time were spoilt by the presence of a number of diesel-engined coaches. The wind remained steady at about 9 m.p.h., from a northerly direction during the course of the experiment, but there were some big changes in ventilation when the fans were switched off during the night.

In each of these experiments samples of smoke were taken, usually over periods of one hour, for the determination of polycyclic hydrocarbons, and in the later series (1959) part of each sample was used for the determination of lead. A continuous record of the carbon monoxide concentration was obtained on two occasions (September 14, 1958 and May 14, 1959) and "spot" measurements were made at other times. Oxides of nitrogen were also measured during some of the experiments, but no observations were made on sulphur dioxide. Since the tunnel air enters from four widely separated points it was considered impracticable to define completely the background level of pollution. All the measurements were made at times of very low background pollution and results from observations in Central London were available for comparison.

Sampling and Analytical Methods

The methods used were similar to those employed in the earlier experiments in bus garages (Commins *et al.*, 1957). Samples of smoke were collected on weighed glass fibre filter sheets measuring 8 in. by 10 in. overall, using a Staplex high volume sampler. Each filter, after weighing, was extracted with cyclohexane for three hours in a Soxhlet apparatus and the yellow extract was reduced to small volume and transferred to a column of alumina. The hydrocarbons were separated chromatographically by elution with cyclohexane and determined spectrophotometrically (Commins, 1958).

Where lead was determined, the filter was divided and a measured fraction of it extracted with nitric acid; an aliquot of this solution was extracted with a small excess of dithizone in chloroform and the excess dithizone removed by washing with ammonia-potassium cyanide solution (Harrold, Meek, and Holden, 1936). The amount of lead in the extract was estimated colorimetrically by a comparison of the red colour of the lead-dithizone complex.

A recording infra-red analyser was used to determine carbon monoxide in the tunnel. Spot samples were collected in evacuated bottles and analysed later in the laboratory.

Oxides of nitrogen were collected by passing the tunnel air through sintered bubblers containing absorbing reagent (Saltzman, 1954). Nitrogen dioxide was absorbed

first and the nitric oxide was then oxidized and collected as NO_2 (Thomas, MacLeod, Robbins, Goettelman, Eldridge, and Rogers, 1956). In the 1959 experiments samples were taken over hourly periods and the colorimetric determinations were made in the tunnel; in some earlier experiments "spot" samples were collected in evacuated bottles and analysed in the laboratory.

Results

The results from each of the experiments carried out during 1958 have been assembled in Table 1. The concentration of "smoke" (including all particulate matter collected by the high volume sampler) ranged from 93 to 235 mg./100 m.^{3}. This is many times the concentration in the ordinary London air at that time of year; samples collected by the same method in Central London at intervals throughout the summer gave results ranging from 10 to 26 mg./100 m.^{3}. To allow some comparison to be made with our earlier bus garage experiments, results from the mid-summer experiments at Merton have been included in this Table. The figures quoted here refer to the period between 11 p.m. and 1 a.m., when the smoke from the buses was sufficient to raise the concentration in the garage to several times the outside level. Under these conditions there was little error in considering the buses to be responsible for all the pollution in the garage, and direct comparisons can be made with the tunnel results. The concentration of each hydrocarbon is much higher in the tunnel than in the garage, but in view of the differences in ventilation little can be inferred from these figures alone. More useful is the comparison of the composition of the smoke, expressed as micrograms of hydrocarbon per gram of smoke. In this respect also the tunnel results are much higher than those obtained in the garage. This suggests that the petrol and diesel vehicles using the tunnel are producing smoke which is richer in hydrocarbons than that emitted by diesel buses when manoeuvring in garages.

The concentrations of pollutants recorded in this series do not vary consistently with the type or amount of traffic, but the smoke concentration was lowest on the Bank Holiday when there were few diesel vehicles present. The concentrations of most of the hydrocarbons at that time were, however, similar to those obtained on other occasions.

The concentration of carbon monoxide observed during the course of these experiments was generally greater than 100 p.p.m. Most of the samples were collected in evacuated bottles over brief periods, but the result quoted for the Rotherhithe Tunnel is an average value over the one and three-quarter hour period of the main experiment. The infra-red analyser was left running for two days before and one day after this experiment. The record showed a further increase in concentration after we had left on the Sunday night, reaching a peak of 500 p.p.m. at midnight. High concentrations were also recorded on the Friday evening, with peak values of 450 p.p.m., and again on Saturday evening, with values up to 340 p.p.m.

Several samples were also collected in evacuated bottles for the determination of oxides of nitrogen, but difficulty was experienced in preventing oxidation of nitric oxide before the determinations were made. Concentrations of nitric oxide ranged from 1 to 8 p.p.m., with smaller quantities of nitrogen dioxide. In later experiments continuous samples were taken and the determinations were made in the tunnel.

Detailed results from the hourly samples taken on May 14, 1959 at point B are given in Table 2. The concentration of smoke varied between 169 and 319 mg./100 m.^{3}, approximately 10 times that in the air of Central London at that time. The concentrations of smoke and of each of the hydrocarbons were in general higher than those encountered in the earlier experiments at the other end of the tunnel (point A). The concentration of carbon monoxide was measured continuously and peak values of up to 235 p.p.m. were recorded. The results for oxides of nitrogen show a much lower ratio of nitrogen dioxide to nitric oxide than is found in the ordinary atmosphere. The concentration of nitric oxide remained close to 1 p.p.m. throughout these experiments, but there was always less than 0·1 p.p.m. of nitrogen dioxide. There was much more lead in the tunnel than occurs normally in urban atmospheres. The concentrations were of the same order of magnitude as those found by Preis (1957) in a road tunnel in Zürich.

In Fig. 2 the relationship between the concentrations of pollutants and the traffic density is shown graphically. All values have been plotted as percentages of the mean for each series so that hour-to-hour variations can be studied on a common basis. The densities of petrol and diesel traffic follow one another fairly closely during the earlier part of the day but in the evening there is a sharp difference between them. The curves have been grouped so as to separate those which show a pronounced rise during the evening from those which do not. The lead and carbon monoxide graphs (Fig. 2) certainly belong to the former category, and follow very closely that for the density of petrol traffic.

Tetra-ethyl lead is added to most petrol, and fine particles containing lead sulphate, lead bromide, and other compounds are emitted with the exhaust gases (Dettling, 1957); diesel fuel does not contain any

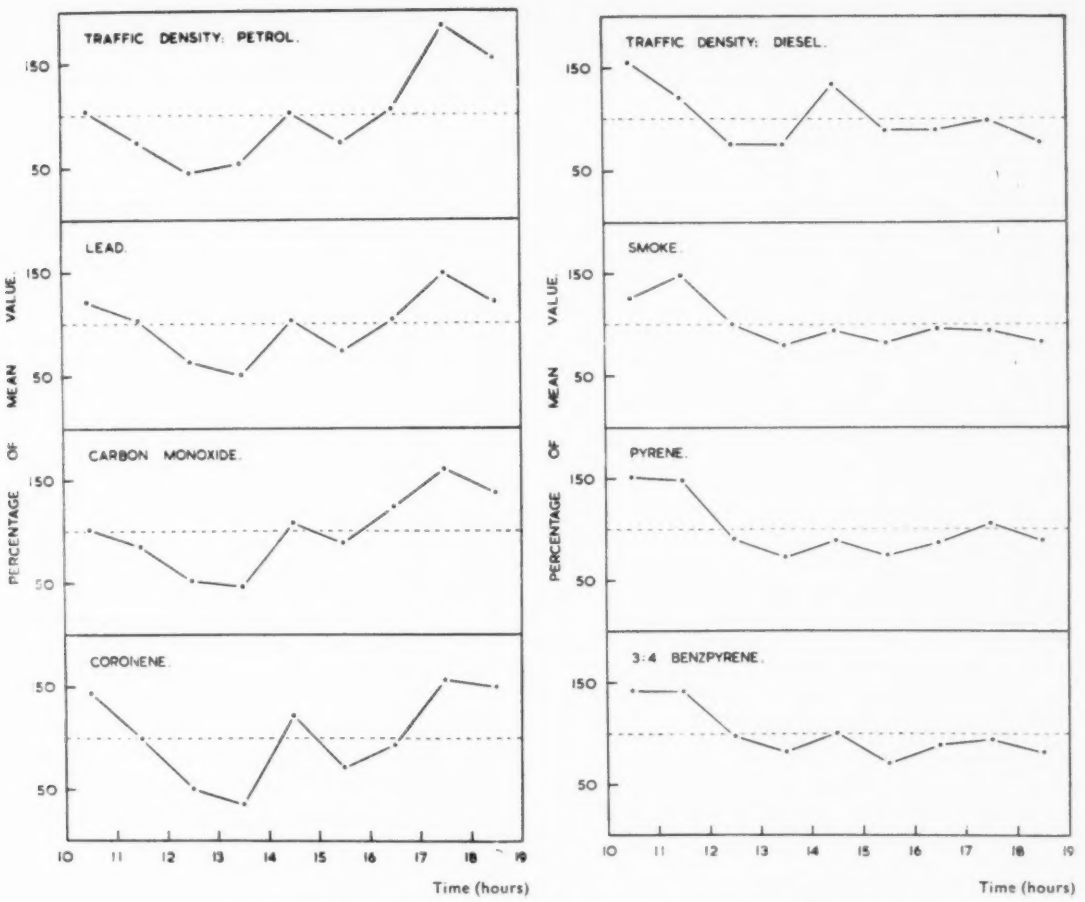


FIG. 2.—Variation in traffic density and concentration of pollutants in Blackwall Tunnel, May 14, 1959.

appreciable quantity of lead. Petrol engines also produce much more carbon monoxide than diesel engines (Fitton, 1958). We would therefore expect the concentrations of lead and carbon monoxide to vary with the amount of petrol traffic, and the similarity between the curves suggests that the density of petrol traffic is in fact a useful index of pollution from petrol-engined vehicles. The curve for coronene has also been included in this series, for it shows some rise in the early evening; the concentration of 1 : 12-benzperylene also varies in a similar fashion.

The concentration of smoke (Fig. 2) does not follow the density of diesel traffic very closely, but the association is certainly closer than with the density of petrol traffic. Since the emission of smoke varies widely with load it may be that the density of the traffic does not provide an adequate index of pollution in the case of diesel vehicles, but it would be difficult to define the operating conditions in the tunnel sufficiently to produce a better one. Variations in the concentration of pyrene and 3 : 4-benzpyrene are also shown in Fig. 2 and the concentrations of fluoranthene and 1 : 2-benzpyrene follow similar patterns. The concentration of anthanthrene varies rather irregularly.

The quality of the smoke can also be considered from the second part of Table 2. The amounts of most of the hydrocarbons (in μg. per gram of smoke) were similar to those found in the earlier experiments, though it can be seen that there is a tendency for the proportions of coronene and 1 : 12-benzperylene in the smoke to increase with the proportion of petrol traffic.

It seems likely from the figures presented so far

TABLE 2

POLLUTION IN BLACKWALL TUNNEL, MAY 14, 1959

	Time (hours)								
	10-	11-	12-	13-	14-	15-	16-	17-	18-19
Traffic flow*: Petrol	702	674	611	631	756	780	1,018	1,241	1,196
Diesel	346	362	326	284	324	311	286	211	194
Speed (m.p.h.)	5·5	7·4	11·1	9·1	6·0	8·7	7·9	5·4	6·3
Traffic density†: Petrol	14·6	10·3	6·3	7·5	14·3	10·2	14·7	26·2	21·6
Diesel	7·2	5·5	3·4	3·4	6·1	4·1	4·1	4·5	3·5
Carbon monoxide (p.p.m.)	76	64	39	35	81	66	92	120	103
Smoke (mg./100 m.³)	268	319	216	169	200	177	204	202	177
Lead (μg./m.³)	23·8	18·7	11·4	9·2	18·8	13·4	19·0	27·2	22·0
Nitric oxide (p.p. 100 m.)	87	133	97	91	137	103	80	121	103
Nitrogen dioxide (p.p. 100 m.)	3	10	7	6	7	7	5	10	7
Hydrocarbons, μg./100 m.³ air:									
Fluoranthene	160	125	109	84	95	81	94	110	79
1 : 2-benzpyrene	24	24	17	14	19	13	17	18	16
Pyrene	139	137	83	66	82	68	80	98	73
Coronene	20	14	7	5	17	10	13	22	21
Anthanthrene	15	26	10	12	9	9	11	7	6
1 : 12-benzperylene	48	40	24	15	41	26	31	51	45
3 : 4-benzpyrene	47	47	32	27	33	23	29	31	27
Hydrocarbons, μg./g. smoke:									
Fluoranthene	597	393	505	495	474	458	465	547	448
1 : 2-benzpyrene	88	75	78	80	94	76	83	89	89
Pyrene	519	430	385	388	408	382	394	487	415
Coronene	75	44	34	27	87	55	64	109	117
Anthanthrene	57	83	48	73	47	50	55	36	35
1 : 12-benzperylene	178	126	111	87	206	148	151	252	252
3 : 4-benzpyrene	176	147	148	157	166	128	142	151	153

*Vehicles per hour in both directions.
†Estimate of number of vehicles in relevant ventilation section (see text)

that both petrol and diesel traffic make some contribution to the concentrations of each of the hydrocarbons determined in the tunnel air, but that the influence of petrol traffic is more pronounced in the case of coronene and 1 : 12-benzperylene than in the others.

Results from the overnight experiment carried out in July 1959 are shown in Table 3. The concentration of smoke varied widely, falling to a minimum of 21 mg./100 m.³ during the middle of the night and reaching a maximum of 182 mg./100 m.³ during the morning rush hour. The mean concentration in Central London at that time was approximately 24 mg./100 m.³. No measurements of carbon monoxide or oxides of nitrogen were made during this series, but the concentrations of lead were determined for each hourly period.

The concentrations of pollutants cannot be related directly to the traffic densities throughout the 14 hours of this experiment, since the ventilation rate was varied during the night. The fans in shafts 2, 3, and 4 were switched off at 23·15 hours as the amount of traffic began to decline, and they were switched on again at 06·30 hours. Those in shaft 1 were switched off at 01·00 hours and on again at 05·30 hours. Around midnight the ventilation still appeared to be satisfactory at the sampling point, even with only one set of fans in operation, but there was a temporary increase in pollution soon after 01·00 hours when all the fans were off. Some brief periods of high pollution were also noticed shortly before all the fans were switched on again in the morning.

Broad comparisons can be made between the first three hours of the experiment and the last three. The density of petrol traffic was similar during these two periods, but there was great variation in the density of diesel traffic. The concentrations of smoke and of each of the hydrocarbons were higher when there were more diesels, and this is in accordance with earlier results, but the concentration of lead was also somewhat increased. Since lead is emitted only by petrol engines this unexpected finding may indicate that there were changes in ventilation due to varying winds or that the intermittent traffic flow during the morning period gave rise to a greater emission of lead per unit petrol vehicle than the smoother flow during the evening.

In general the smoke from this series contained smaller amounts of hydrocarbons (expressed as μg. per g.) than that collected in the earlier daytime experiment. This means either that the vehicles were emitting material which was weaker in hydrocarbons than before, or that a greater amount of extraneous dust was being picked up on the second occasion.

Particle Size Distribution of the Smoke

During the course of the study observations were being made on the size and shape of particles in the atmosphere of London, and a number of samples were collected for this purpose in the Blackwall Tunnel. The samples were collected on formvar films in a thermal precipitator and examined on an electron microscope. Nearly all the particles were small smoke aggregates, with a mass median diameter of approximately 1 μ (Waller, Brooks, and Cartwright, 1961). The form of the particles was typical of those produced by the incomplete combustion of hydrocarbon fuels. All were small enough to stay in suspension indefinitely and they were well within the respirable size range.

Discussion

The primary object of this investigation was to assess the magnitude of the problem of air pollution by motor traffic and in order to do this we worked in the most polluted places we could find. In these two tunnels we were able to study pollution by petrol and diesel vehicles in widely varying mechanical states and operating conditions, and the main object of the study has been achieved. As our work progressed we began to hope that by comparing the pollution during selected periods of diverse traffic composition we might be able roughly to identify the main sources, petrol or diesel, of individual pollutants. In this secondary aim we were only partially successful and the apparent correlations between the concentrations of various pollutants and the type and amount of traffic must be interpreted with great caution. The Blackwall Tunnel has two bends which tend to impede large vehicles (which are mostly diesel powered) and thus radically alter the flow of traffic; a curious consequence of this impedance is that large diesel vehicles can seem to emit lead by holding up petrol traffic. This obviously false relationship between the nature of traffic and one pollutant serves to sound a note of caution when we consider the emission of other pollutants, the source of which is not known with the same certainty. No account has been taken of variations in types of vehicle within the general categories of "petrol" and "diesel"; during the daytime there is a large proportion of commercial traffic and the emissions from diesel or petrol engined lorries may differ from those of the buses or cars which make up the bulk of the traffic at other times. Over a limited period however the concentrations of both lead and carbon monoxide in the tunnel air vary with the amount of traffic, and the results from the experiment carried out on May 14, 1959 show a real relation between the concentrations of these pollutants and the density of petrol traffic, which must then be considered as an important source of carbon monoxide and lead compounds. Industrial

TABLE 3

POLLUTION IN BLACKWALL TUNNEL, JULY 5-6, 1959

	Time (hours)													
	20-	21-	22-	23-	24-	1-	2-	3-	4-	5-	6-	7-	8-	9-10
Traffic flow*:														
Petrol	1,265	1,321	1,481	1,292	632	155	34	30	40	197	618	1,072	1,006	838
Diesel	49	49	39	27	24	17	7	11	14	76	221	327	367	335
Speed (m.p.h.)	9·5	10·5	17·8	18·6	22·7	29·2	29·2	21·5	24·1	21·5	17·0	6·1	10·2	6·1
Traffic density†:														
Petrol	15·1	4·31	9·5	7·9	3·2	0·6	0·13	0·16	0·19	1·04	4·12	20·0	11·2	15·6
Diesel	0·59	0·53	0·25	0·17	0·12	0·07	0·03	0·07	0·07	0·40	1·47	6·09	4·08	6·24
Smoke (mg./100 m.³)	46	59	45	44	32	41		21	24	55	112	147	182	159
Lead (μg./m.³)	11	17	11	14	8	15		3	5	6	11	21	20	22
Hydrocarbons μg./100 m.³ *air:*														
Fluoranthene	7·9	17·3	11·2	3·9	3·9	7·6		2·8	1·5	1·4	17·4	35·1	40·0	34·6
1 : 2-benzpyrene	3·6	6·7	4·7	2·5	2·7	2·9		0·8	1·1	0·8	5·6	10·7	11·7	12·5
Pyrene	6·4	13·6	8·5	3·7	2·9	7·4		1·9	1·2	1·2	15·6	37·1	38·4	35·8
Coronene	5·7	8·2	3·8	3·9	3·3	3·9		0·6	1·3	0·9	4·1	14·6	16·7	19·3
Anthanthrene	0·8	4·4	2·1	1·3	1·2	3·2		0·3	0·6	0·2	2·7	9·8	10·2	11·5
1 : 12-benzperylene	9·7	17·4	9·5	8·9	7·2	9·7		1·8	3·2	2·0	11·7	35·5	36·6	41·6
3 : 4-benzpyrene	5·8	13·5	8·0	3·9	4·4	4·5		1·5	1·6	1·5	7·8	13·8	17·7	16·5
Hydrocarbons μg./g. *smoke:*														
Fluoranthene	172	290	253	89	121	185		134	64	26	156	240	19	216
1 : 2-benzpyrene	78	113	106	57	83	71		40	45	15	50	73	264	78
Pyrene	137	229	192	83	89	179		94	48	21	140	253	211	224
Coronene	124	138	86	88	103	94		29	55	16	36	100	91	121
Anthanthrene	17	73	47	29	37	56		14	26	4	24	67	56	72
1 : 12-benzperylene	210	293	216	201	224	236		90	133	36	105	242	207	260
3 : 4-benzpyrene	126	227	181	87	135	110		75	68	27	70	94	97	103

*Vehicles per hour in both directions.
†Estimate of number of vehicles in relevant ventilation section (see text).

TABLE 4

POLLUTION IN BLACKWALL TUNNEL, SUMMARY OF RESULTS

	Blackwall Tunnel					Central London		
	Morning Rush Hour	Evening Rush Hour	Daytime	Holiday Evening	Night Minimum	Typical Summer	Typical Winter	Temperature Inversions
Petrol vehicles per hour	921	1,129	692	1,454	30	—	—	—
Diesel vehicles per hour	306	212	326	50	11	—	—	—
Speed (m.p.h.)	5	4	8	9	22	—	—	—
Smoke (mg./100 m.³)	178	166	225	72	21	20	35	670
Lead (μg./m.³)	21	23	16	13	3	1	2	5
Carbon monoxide (p.p.m.)	132	105	60	138	—	17*	11*	360†
Hydrocarbons (μg./100 m.³)								
Fluoranthene	46	62	109	27	2·8	1·0	2·5	407
1 : 2-benzpyrene	14	14	18	8	0·8	0·8	2·0	74
Pyrene	64	64	96	35	1·9	1·0	2·5	317
Coronene	24	22	12	14	0·6	0·4	1·0	28
Anthanthrene	12	10	14	8	0·3	0·2	0·5	38
1 : 12-benzperylene	43	40	32	24	1·8	0·8	2·5	106
3 : 4-benzpyrene	20	23	35	14	1·5	1·2	5·0	222

*Mean of street concentrations reported by Wilkins (1956).
†Highest value in series of instantaneous samples taken in busy streets.

and domestic chimneys also emit carbon monoxide and small quantities of lead but these sources are unlikely to contribute significantly to the concentrations occurring amongst heavy traffic. Brief, Jones, and Yoder (1960) have reported close correlations between the concentrations of these pollutants and the amount of traffic in the streets of a north-eastern United States city. These authors did not distinguish between petrol and diesel traffic, but the latter probably formed only a small percentage of the total. In Central London a substantial proportion of the traffic has diesel engines which can be discounted as emitters of lead and carbon monoxide. Our work in streets, garages, and tunnels leads us to support the suggestion of Wilkins (1956) that the current use of diesel engines in buses and heavy goods vehicles provides one explanation for the lack of any substantial increase in carbon monoxide concentrations in London streets over a period of 24 years, despite large increases in traffic.

The relationship seen in our experiments between the concentration of smoke in the tunnel and the density of diesel traffic also seems reasonable. Williams (1961) has shown that "the smoke concentrations from road traffic on busy roads can be appreciable and, for short periods, can produce intense local pollution". In Table 4 the tunnel results have been summarized and compared with outdoor concentrations of smoke and hydrocarbons. The lowest pollution encountered in the tunnel has been tabulated together with the mean values for other periods; the morning "rush hour" figures refer to samples taken between 07·00 and 10·00 hours on week-days, the evening "rush hour" to samples taken between 16·00 and 20·00 hours, and the daytime figures to those taken between 10·00 and 16·00 hours. The "holiday evening" samples were taken in the Blackwall Tunnel on a Sunday or Bank Holiday. The Central London figures provided for comparison are, with the exception of the carbon monoxide, based on results from a number of samples taken in the open air, away from traffic, at St. Bartholomew's Hospital. Typical summer and winter values are quoted together with the results from a sample taken during a period of high pollution on December 3, 1957; the smoke on that occasion came mainly from domestic chimneys and a low temperature inversion prevented it from dispersing normally.

During the quietest period in the middle of the night the concentrations of most pollutants in the tunnel fall to the same order of magnitude as those in the ordinary London air. At other times, when there is heavy traffic, the concentrations of all pollutants measured in this study are much higher than the typical summer or winter levels in outdoor air. The concentrations during the morning and evening rush hours are similar to one another, despite differences in traffic composition. The maximum concentration of smoke occurs in the daytime, when the number of diesel vehicles is highest. The concentrations of most of the hydrocarbons are also highest then, but coronene and 1 : 12-benzperylene, which appear to be associated rather more with petrol vehicles, show their highest concentrations during the rush hours. The concentration of carbon monoxide is highest on holiday evenings, when the number of petrol vehicles is at a maximum. The concentration of smoke and of most hydrocarbons is lower then than at any other busy time in the tunnel, although it is still many times the outdoor level.

Further comparisons within Table 4 show that all the concentrations of smoke and hydrocarbons in the tunnel are within the range found in ordinary London air. Those observed during busy periods can be regarded as similar to the concentrations of smoke and hydrocarbons found during temperature inversions on winter evenings in Central London. Carbon monoxide appears to be the only significant hazard; during the rush hours and on busy holiday evenings the concentration was generally over 100 p.p.m., which is the maximum allowed for continuous exposure in industry. Exposure to these concentrations for several hours may have perceptible effects and on several occasions during the course of these experiments the authors and their colleagues experienced headaches, fatigue, and general drowsiness after being exposed continuously for periods of three to 14 hours in the tunnel. Drivers are not normally exposed to these concentrations for more than 30 minutes in passing through the tunnel and no one else spends long there during the busy periods. Severe eye irritation was experienced at times, but the cause of this was not investigated. The oxides of nitrogen do not appear to present any special hazard. There is much more nitric oxide than nitrogen dioxide in the tunnel air. The concentration of the latter reached about 0·1 p.p.m. (Table 2), which is well below the 5 p.p.m. permitted in industrial atmospheres. The physiological effects of nitric oxide have not been studied widely, but it can combine with the blood in the same way as carbon monoxide. The effects of oxides of nitrogen have been discussed by Katz, Rennie, and Jegier (1959), who made measurements in a railway tunnel where diesel trains were operating; they considered that the concentrations of nitric oxide observed there (of the order of 20 p.p.m. in instantaneous samples) were not sufficient to form detectable amounts of methaemoglobin in blood. There is at present no reason to believe that the concentrations recorded in the tunnel (up to 1·4 p.p.m. in continuous estimations, but higher in some spot samples) would produce any effect on man.

Conditions in the Blackwall Tunnel are expected to improve greatly when the twin tunnel now under construction comes into use. The existing tunnel will then be used for traffic in one direction only.

Valuable assistance was given by Mrs. R. C. Hogan, Mrs. J. V. Dale, and other members of the Air Pollution Research Unit. We are indebted to the London County Council for providing facilities for this investigation.

References

Brief, R. S., Jones, A. R., and Yoder, J. D. (1960). *J. Air Pollution Control Ass.*, **10**, 384.
Commins, B. T. (1958). *Analyst*, **83**, 386.
——, Waller, R. E., and Lawther, P. J. (1957). *Brit. J. industr. Med.*, **14**, 232.
Dettling, J. (1957). *Z. Präv.-Med.*, **2**, 140.
Fitton, A. (1958). *Proceedings of the Conference on the Mechanical Engineer's Contribution to Clean Air*, p. 151. Institution of Mechanical Engineers, London, 1957.
Harrold, G. C., Meek, S. F., and Holden, F. R. (1936). *J. industr. Hyg.*, **18**, 724.
Katz, M., Rennie, R. P., and Jegier, Z. (1959). *A.M.A. Arch. industr. Hlth*, **20**, 493.
Preis, H. (1957). *Z. Präv.-Med.*, **2**, 397.
Regan, C. J. (1932). London County Council. Annual Report of the Council, 1930, Vol. 4, (Part 3), p. 271.
Saltzman, B. E. (1954). *Analyt. Chem.*, **26**, 1949.
Thomas, M. D., MacLeod, J. A., Robbins, R. C., Goettelman, R. C., Eldridge, R. W., and Rogers, L. H. (1956). *Ibid*, **28**, 1810.
Waller, R. E., Brooks, A. G. F., and Cartwright, J. (1961). To be published.
Wilkins, E. T. (1956). *Roy. Soc. Hlth J.*, **76**, 677.
Williams, F. P. (1961). *Revue Trimestrielle de l'Association pour la Prévention de la Pollution atmosphérique*, **3**, 595.

Brit. J. industr. Med., 1961, **18**, 260.

A CLINICAL STUDY OF ELECTRICAL ACCIDENTS

BY

W. R. LEE

From the Nuffield Department of Occupational Health, University of Manchester

(RECEIVED FOR PUBLICATION MAY 18, 1961)

The report of a clinical study of 104 electrical accidents which befell 85 men is divided into two parts.

Part I enumerates the different types of accidents as flash burn, Joule burn, arc eye, "held on" shock and "not held" shock, physical shock, and death. These are related to the different voltages involved ranging from 240/415 (medium) voltage to 33 kilovolts. There appeared to be no association between voltage and type of injury and no evidence to suggest that any of the voltages are free from hazard. There were 53 cases of flash burn, affecting mostly the face and extensor surface of the hands and arms. The 16 cases of arc eye caused no serious concern. Of the 15 Joule burns all except one occurred at medium voltages in "held on" accidents, the other being associated with an electric shock at 33 kilovolts. The majority of Joule burns affected the flexor surfaces.

Part II of the paper deals with the 43 cases of electric shock (passage of current through the body). Thirty of these cases were "held on" to the circuit by the current. It was found that the longer a victim was held on to the circuit the greater appeared to be his chances of developing heart and chest symptoms suggestive of impending asphyxia, and of losing consciousness. Although about half of these men were released by an external agency and others struggled off, a number suddenly became free from the circuit without, they claimed, losing consciousness. This is difficult to explain. Artificial respiration was administered in two cases, one of whom was "held on" and was being asphyxiated. The other case received flash burns only and did not in fact receive an electric shock.

PART I: GENERAL REVIEW AND NON-SHOCK CASES

Electrical accidents receive little attention in British medical literature. In fact, in the electricity supply industry they account for only 1% of the total number of accidents but they are the commonest cause of accidental death (Hughes and Corney, 1956). In factories, electrical accidents account for less than 0·5% of all accidents, but more than 5% of these electrical accidents prove fatal (Emerson, 1961). This paper reports a clinical investigation into a series of electrical accidents; detailed consideration of the cases of electric shock (passage of current through the body) is given in Part II of this paper.

Method

The names of all men in an electricity supply undertaking who had sustained an electrical accident during the preceding three years were obtained. Of the total of 114, 107 were still employed by the undertaking. One man was killed; the remainder were contacted and 84 attended for interview. (The majority of those who were not seen worked in one small area and there is no reason to suppose that the nature of their accidents was any different from the whole.) They were all seen by one investigator and were questioned about the electrical and physical circumstances of the accident, about symptoms, and about subsequent medical history. Parts of the body reported as injured in the accident were examined and in cases of electric shock a complete physical examination was carried out. In addition to the electrical accidents for which they were seen (referred to hereafter as "series cases"), these persons recounted a further 19 electrical accidents (referred to hereafter as "additional cases"). It must be acknowledged that whilst the 84 "series cases" represent a group of electrical accidents selected only in the sense of those attending, the 19 "additional cases" are biased in content as a result of the investigator's interest in a particular type of electrical accident. For this reason "series cases" are considered separately from "additional cases" when the incidence of injuries is discussed.

TABLE 1
INCIDENCE OF INJURIES

	Flash Burn	Joule Burn	Arc Eye	Shock "Held On"	Shock "Not Held"	Physical Shock	Death	No. of Accidents*
Series cases	53	10	16	14	10	3	1	85
Additional cases	1	5	—	16	3	1	—	19
Total	54	15	16	30	13	4	1	104

*Several subjects sustained more than one class of injury as the result of a single accident.

TABLE 2
VOLTAGE INVOLVED

	Medium	2·2-2·4 kV	6·6 kV	11 kV	33 kV	Total
Series cases	69	3	3	7	3	85
Additional cases	18	—	—	1	—	19
Total	87	3	3	8	3	104

TABLE 3
RELATIONSHIP BETWEEN INJURY AND VOLTAGE (SERIES CASES)

Voltage	Flash Burn	Joule Burn	Arc Eye	Shock "Held On"	Shock "Not Held"	Physical Shock	Death	No. of Accidents*
Medium	43	8	14	13	9	1	—	69
2·2-2·4 kV	3	—	—	—	—	—	—	3
6·6 kV	2	1	—	1	—	—	—	3
11 kV	3	—	1	—	1	3	—	7
33 kV	2	1	1	—	1	—	1	3
Total	53	10	16	14	11	4	1	85

*Several subjects sustained more than one class of injury as the result of a single accident.

Electrical Accidents

Electricity can cause injury to the body in three ways. First, by the conversion of electrical energy into light energy, which may injure the eye. Second, by the conversion of electrical energy into heat energy. If this takes place outside the body the heat is usually associated with a short-lived flame causing a flash burn. Whereas if the conversion of electrical energy into heat takes place within the body most heat is evolved where the resistance is highest, in conformity with Joule's Law, usually where the electrodes are in contact. The resulting burn is frequently referred to as a Joule burn. Third, the passage of an electric current through the body produces other effects grouped under the term "electric shock". In this study, cases of electric shock have been divided into two groups according to whether or not the victim was "held on" to the circuit by the current. The cases of electric shock are considered in greater detail in Part II of this paper. Finally, as in any accident, effects might be produced which are not the results of any of the electro-pathological phenomena. These are grouped under the term "physical shock", and are considered with the cases of electric shock.

Incidence

Table 1 shows the incidence of injuries in both the "series cases" and "additional cases". It will be seen that burns account for at least half of the injuries.

Voltage

Electricity is usually received by the distribution undertakings at 33 kV and transmitted through their networks at this voltage and at 11 kV, 6·6 kV, and 2·4 kV. Finally, the voltage is reduced to 415/230 (the so-called medium voltage) for distribution to the majority of consumers. It can be seen from Table 2 that in the "series cases" accidents occurred at all these voltages. It is not possible to draw conclusions regarding the proportions of accidents at different voltages, for the number of man-hours exposed at these voltages is not known. Certainly it is seen that the medium voltages are not free from hazard. Emerson (1961) states that in Great Britain approximately two-thirds of the electrical fatalities occur at voltages below 250.

The relationship between injury sustained and voltage involved, in the "series cases" only, is shown in Table 3. It is seen that there is no indication that

any particular voltage is associated with certain types of injury and, more important, there is no evidence to suggest that any of the voltages are free from hazard. All but one of the 14 "held on" cases occurred at medium voltage; one occurred at 6,000 volts. Of the 16 "held on" accidents seen in the "additional cases" all occurred at medium voltage. Whilst it is accepted that the phenomenon of being "held on" to a circuit is dependent on the strength of a current rather than its voltage (Dalziel, Lagen, and Thurston, 1941), it is of interest to note that "held on" shocks are generally associated with medium voltages.

Arc Eye

The occurrence of this condition following the flash of a short-circuiting current was first described by Rivers (1894) and Roy (1897). Altogether 16 subjects described the typical symptoms of arc eye, appearing a few hours after a flash accident. Nearly all these people were exposed to a short-lived flash within one or two feet of the eyes; in fact in seven of them there was singeing of the eyelashes, eyebrows, or hair, or first degree burns of the face.

In one case the arc eye was accompanied by other more serious injuries (second degree burns of the hands) which occasioned considerable loss of time from work (this was the same accident in which the only death in the series occurred). Omitting this case the 15 other cases of arc eye lost only 22 days from work between them as a result of the accident. The longest period off work was seven days, whilst nine persons lost no time at all. No cases of the less common, but more lasting forms of eye injury, such as the retinal changes described by Minton (1949), were encountered.

Burns

Flash Burns.—As had been stated, flash burns result from a flame of very short duration. It is therefore to be expected that the exposed parts of the body, namely the face and extensor surfaces of the hands and arms, would be affected most frequently. It was found that 28 flash burn accidents affected the face, 37 the extensor aspects of the arms and hands, and only 10 the flexor aspects; two were unspecified (the total of these exceeds the total number of flash burn accidents (54) as an accident frequently resulted in burns at more than one site).

The periods of time lost as a result of flash burn are shown in Table 4.

It will be seen that just over half the flash burn accidents reported resulted in no lost time and that 42 out of 54 of the cases were back at work in a month. Five cases (just less than 10%) were off for more than three months, and three of these were off for six months or longer.

Case 6.—He sustained second degree burns on the backs of both hands and a first degree burn on the left side of the face when arcing occurred at 415 volts at a street pillar on which he was working. As a result of the accident the pillar was destroyed and the patient's suit burned although it did not catch fire. He was still off work when seen eight months later.

Case 7.—This man was working on a 33,000 volt line in a sub-station when there was an explosion killing his colleague. In addition to arc eye and severe second degree flash burns of the face and backs of hands he sustained Joule burns of both palms and a violent electric shock which threw him off. He was away from work for just over six months.

Case 51A.—This electrician was screwing a metal plate into position. The plate became live at 230 volts and flashed across to the neutral conductor. He estimated that the arcing lasted about two minutes, until the metal ends had burned themselves away. He states that he was held on to the circuit and close to the burning (although his left hand was holding the live metal plate, his right hand held an insulated screwdriver and he wore dry substantial rubber boots). This accident was in the "additional cases" series and had occurred several years previously; it was not possible to check details from the safety officer, *e.g.* the efficiency of insulation of the screwdriver. He was off work for about three months.

Case 58.—This man was injured during a flashover explosion between two 11,000 volt lines. He sustained burns to the backs of the hands and the front of the face and neck, which resulted in his being off work for about three months.

Case 75.—A jointer was working in a sub-station when there was an explosion in a nearby 6,600 volt apparatus to his right. The flame burned the backs of both his hands, his face, and the front of his scalp. His trousers and overall collar caught fire and resulted in burns to the neck and legs. The complication of flame burning by ignition of clothing has been discussed by Tempest and Atkins (1958). As a result of this accident the man was away from work for about six months.

It will be seen that out of five flash burn accidents resulting in absences of six months or longer, three occurred at voltages greater than 6,000. The other two occurred at medium voltage and in one of these the subject was apparently "held on" close to the burning.

Joule Burns

Voltage and duration of current.—A Joule burn is caused by the passage of electric current through the skin. The heating effect of a current is given by the formula: I^2Rt where I = current, R = resistance, t = time.

For a given resistance, medium voltages are associated with smaller currents than higher voltages.

TABLE 4

TIME LOST FROM FLASH BURNS

Nil	< One Week	One Week to One Month	One to Three Months	> Three Months	Total
29	5	8	7	5	54

It can, therefore, be expected that Joule burns would be caused by medium voltages only if the victim remained in contact with the conductor, whereas a shock of high voltage (likely to be associated with a greater current) might cause burning even if the contact were only brief.

In fact out of 15 cases of Joule burns reported, 14 were associated with "held on" accidents at medium voltage, the other case being associated with an electric shock at 33,000 volts.

As described in Part II of this paper, victims of "held on" accidents were asked to estimate the length of time for which they were held on. On this basis they were divided into three broad groups. But there is no indication that those held on for longer periods were more liable to Joule burn (Table 5). It is possible that whereas the occurrence of Joule burns was not related to the length of time a victim was held on, the severity might have been. The method of investigation in which men were interrogated, in the "additional" cases, several years after the incident, made it impossible to assess the severity of Joule burns, except perhaps by consequent sickness absence. In view of the small number (only 15 cases of Joule burn) it was not feasible to use this method.

Site.—Because a Joule burn results from contact of the body with a conductor this type of accident would be expected to produce injury to the flexor aspect of the fingers or palms and in contrast to flash burns the face would escape. Thirteen Joule burns were on the flexor aspects of the hands or arms and two were on the extensor aspects. One of these burns was on the left thigh and one on the side of the neck.

The case of Joule burning of the side of the neck merits comment.

CASE 69A.—An electrician was standing on a pair of wooden steps holding an earth wire in the left hand whilst stapling it to a beam. He intended to join it to a second wire which was connected to earth. On grasping the second wire with the right hand he received a violent electric shock and was unable to let go (subsequently it proved that the first earth wire which he had been stapling was energized to 240 volts). During the violent shaking which affected him during the shock the energized wire fell across the left side of his neck, where he developed a linear burn. There were no Joule burns on the hands.

It is not easy at first to see why he should develop a burn on the neck and not on the hand. Certainly the skin of the neck is of finer texture than that of the hand and presents a lower resistance to the passage of a current. From the formula (I^2Rt) quoted earlier, this means that less heat is released. But for a given voltage the current will increase if the resistance is lowered (Ohm's Law), therefore the increased current would cause burning. However, if this were so, the greater current would also pass through the right hand, where the heating effect would be greater than at the neck (for the resistance of the skin of the hand is greater). A possible explanation of the phenomenon is that the area of contact of the conductor at the neck was considerably less than at the right hand and hence the current density (and thus localization of the heating) was greater.

TABLE 6

RELATIONSHIP OF JOULE BURN WITH CURRENT PATHWAY

	Arm to Arm	Arm to Leg	Total
Joule burn	8	6	14
No Joule burn	7	9	16
Total	15	15	30

Current Pathway.—In cases of "held on" shock no association was found between the path which the current is presumed to have taken through the body and the incidence of Joule burns (Table 6).

TABLE 5

RELATIONSHIP OF PERIOD "HELD ON" AND JOULE BURN

	Few Seconds	30 to 60 Seconds	One to Four Minutes	Unknown	Total
Joule burn	7	3	4	—	14
No Joule burn	6	2	6	2	16

Conclusion

In this series of electrical accidents there were 53 cases of flash burns, 25 cases of shock (including "held on" and not held), and 16 cases of arc eye. There appeared to be no association between voltage and type of injury. The cases of arc eye caused no serious concern. The cases of flash burns varied from trivial singeing of the hairs on the face to more serious burns of hands and face. It seems, as might be expected, that burns from higher voltage flashes are more likely to cause lost time than burns from medium voltage flashes. One case of Joule burn was associated with a high voltage shock, all the others were associated with "held on" type of accidents at medium voltage. In contrast to the flash burns the majority of Joule burns occurred on the flexor surfaces.

Although this series was drawn from persons dealing with high and medium voltages, it is pertinent to note that the medium voltages to which the general population is exposed can be responsible for all the types of hazards encountered.

PART II: ELECTRIC SHOCK

Part I of this paper describes the method of investigation of a number of victims of electrical accidents and discusses those accidents which did not result in passage of an electric current through the body. This part of the paper considers the cases in which the current did pass through the body. These cases of electric shock are divided into two groups (the "not held" and the "held on") depending on whether or not the victim was held on to the circuit by the current. The three cases of "physical shock" mentioned in the previous paper are also considered.

"Not Held" Shock

There were 13 cases in this group, of whom 10 were "series cases" and three were "additional cases". They ranged from the severe, to the moderate, when judged by their immediate effects. Two cases in which there was some doubt as to whether or not shock had occurred have been included, but because of the possibility of electric shock they could not be included in the physical shock cases.

The following is an example of the severe type.

Case 13.—A 60-year-old labourer, wearing rubber boots, was standing in a damp trench sawing through a cable believed to be dead. In fact, the cable was energized to 11,000 volts. There was a sudden flash and he became rigid and was thrown backward into the trench; the saw was destroyed. He felt very dazed and although able to climb into the ambulance he did not really recover consciousness until he arrived in hospital a few minutes later. He was detained only a few hours and then sent home. As a result of the accident the backs of his hands were scorched, but not badly enough to require dressing. He was off work only for one day.

As he was wearing rubber boots it is considered more likely that the path of the current was from right hand to left hand (his left hand was holding the earthed armoured covering of the cable) than from hands to feet. He was not wearing gloves.

The moderate type of shock is more familiar.

Case 81.—A 54-year-old female assistant cook was cleaning the top of an electric cooker. She was wearing rubber shoes, she had her left hand on the top of the cooker and a wet cloth in her right hand. She felt a shock up her right arm and the hand contracted breaking the circuit immediately. She was quite well immediately after and lost no time from work. This was presumably a brief arm-to-arm shock.

The two cases in which it was doubtful if a shock had been received are detailed.

Case 25.—A 21-year-old clerk was putting a plug into a metal clad 230 volt socket, in which, apparently, the live wire had become loose and was touching the metal casing. He pressed the switch with his left hand and there was a flash, flame escaping through the chipped plug top. He stated that he felt a slight shock up his left arm. His right arm was not in contact with anything and he was wearing rubber-soled shoes and standing on a wooden floor. If he received a shock it was presumably from the left arm to the feet. This is feasible, for according to Dalziel *et al.* (1941) the threshold of perception is 1mA. Applying Ohm's law this allows for a resistance to earth of 230,000 ohms; the resistance of the body could account for anything from 1,000 to 500,000 ohms (Dalziel, 1947) or 800 to 100,000 ohms (Kouwenhoven, 1949), although Emerson (1961) quotes values of 200 to 500 ohms.

Case 32.—A 41-year-old electrician was attending to a domestic immersion heater which was believed to be dead. He grasped two wires with the right hand which were in fact the live and earth conductors. There was a flash and the fuse blew. It is not clear whether the current flowed from one part of his hand to the other or if the two wires touched. There was a very slight burning of the skin, not sufficient to cause blistering. The man was able to carry on with the job and lost no time from work. The question of an electric shock occurring when the two conductors are in contact with the same limb has been mentioned by Kouwenhoven (1949) who reports cases of loss of consciousness from this type of shock even when there has been no burning of the skin.

"Held On" Shock

There were 30 accidents in which the patient reported that he had been held on to the circuit by the current. The question of Joule burning in these

circumstances was discussed in the first part of this paper.

Chest and Heart Symptoms.—An interesting feature of the "held on" accidents was the occurrence of chest and heart symptoms, whilst the victim was being held on to the circuit. Ten persons reported chest symptoms such as tightness around the chest, difficulty in breathing, and a sensation of strangulation. Symptoms related to the heart action, such as marked palpitation and a feeling of the heart pounding strongly against the chest wall, were noticed by six persons. Some cases suffered from both chest and heart symptoms.

Joule Burns.—It could be argued that if the skin resistance is lowered as a result of a Joule burn then the consequent increase in current passing through the body would be more likely to produce heart and chest symptoms.

The effect of Joule burning on the resistance of the skin is not simple. Oliver (1913) and François (1955) have stated that there is an increase in resistance, in severe burning. Whereas Kouwenhoven (1949) considers that blistering of the skin in Joule burning leads to a lowered resistance, Lewis (1958) states, "when the skin is burned its resistance is greatly increased; this may break the current. If carbonization takes place resistance of the skin is reduced". It will be seen from Tables 7 and 8 that no association between Joule burning and the reporting of heart and chest symptoms was observed.

Pathway of Current.—Ferris, King, Spence, and Williams (1936) showed that in sheep the minimum current required to produce ventricular fibrillation was 23 mA when passed from foreleg to hindleg and 136 mA when passed from foreleg to foreleg. Incidentally, Dalziel (1946) in a critical study of these findings concluded that the minimum fibrillating current for men was about 100 mA. Kouwenhoven, Hooker, and Langworthy (1932) measured, by means of a ring transformer, the proportion of the total current which passed through the heart of experimental animals. They found that 7% of a current passing from upper extremity to lower extremity went through the heart, whereas if the pathway was from foreleg to foreleg only 3% passed through the heart. They concluded that "as far as the heart is concerned, fibrillation will be produced by a much smaller total current flowing from the upper to the lower extremities than between the forelegs" and continued, "In most industrial accidents the current flows from the right hand to the feet and thus the heart carries a greater proportion of total current than when contact is made with the circuit at any other locations".

TABLE 7

RELATIONSHIP OF JOULE BURN WITH CHEST SYMPTOMS

	Joule Burn	No Joule Burn	Total
Chest symptoms	4	7	11
No chest symptoms	10	9	19
Total	14	16	30

TABLE 8

RELATIONSHIP OF JOULE BURN WITH HEART SYMPTOMS

	Joule Burn	No Joule Burn	Total
Heart symptoms	2	4	6
No heart symptoms	12	12	24
Total	14	16	30

TABLE 9

RELATIONSHIP OF PATHWAY WITH HEART SYMPTOMS

	Arm to Arm	Arm to Leg	Total
Heart symptoms	2	4	6
No heart symptoms	13	11	24
Total	15	15	30

TABLE 10

RELATIONSHIP OF PATHWAY WITH CHEST SYMPTOMS

	Arm to Arm	Arm to Leg	Total
Chest symptoms	5	6	11
No chest symptoms	10	9	19
Total	15	15	30

Table 9 shows that in the present series there is no marked association between pathway and heart symptoms.

A similar result is found when the relationship between pathway and chest symptoms is examined (Table 10).

Relationship between Chest Symptoms and Heart Symptoms.—It has been shown by Dalziel *et al.* (1941) that persons were unable to let go of a conductor carrying a 60 c.p.s. alternating current exceeding 9·7 to 21·6 mA (all the subjects could release at the lower value, only one at the higher). The difference in frequencies between British 50 c.p.s. and U.S.A. 60 c.p.s. does not materially affect these figures (Dalziel, Ogden, and Abbott, 1943). As noted earlier the threshold for ventricular fibrillation in man is believed to be about 100 mA and it is to be expected that the current necessary to produce more forceful heart action will be less (by an

TABLE 11

RELATIONSHIP OF HEART SYMPTOMS WITH CHEST SYMPTOMS

	Chest Symptoms	No Chest Symptoms	Total
Heart symptoms	4	2	6
No heart symptoms	7	17	24
Total	11	19	30

unknown amount). Nevertheless, it might be interesting to see whether persons presented a higher incidence of heart symptoms when subjected to a current which, although great enough to cause chest symptoms, was obviously not sufficient to cause fibrillation. The results are given in Table 11.

It is seen that out of 11 patients with chest symptoms four had heart symptoms as well, but out of the 19 subjects in which the current was insufficient to cause chest symptoms only two had heart symptoms. These figures only suggest an association between a current strong enough to cause impending asphyxia and the occurrence of heart symptoms. The explanation could lie in increased heart beat caused by the impending asphyxia (Bell, Davidson, and Scarborough, 1956) or a psychological reaction induced by the fear of being held on.

Period of time held on.—It is very difficult for a person, who has undergone the terrifying experience of being held on to an electrical circuit, to estimate accurately the length of time for which he had been held. Nevertheless, all except two men were prepared to estimate this period. It was expressed vaguely and varied from a "few seconds" to "about four minutes". For the purpose of this study it has been decided to make a classification by three periods; "a few seconds", "minutes", and an intermediate group called "30 to 60 seconds". The relationship between period held on and other factors has been studied.

It was noticed in Part I that persons held on for longer periods did not seem any more likely to suffer from Joule burns.

However, chest symptoms were more frequent in persons held on for longer periods, as is seen in Table 12.

Out of 13 persons held on for a few seconds, only two developed chest symptoms whereas of 10 persons held on for minutes, six developed these symptoms.

A similar trend is seen with regard to heart symptoms although the figures are too small to be conclusive (Table 13).

Loss of consciousness.—Of the 30 men who were "held on", eight reported that they lost consciousness. (Many of these were able to fill in details of the accident from what they had subsequently been told.) It is apparent from Table 14 that those men held on for longer periods are more likely to lose consciousness.

Thus, it will be seen that the longer a victim is held on to an electrical circuit the greater appear to be his chances of developing chest symptoms (indicative of developing asphyxia), possibly heart symptoms, and of losing consciousness. As the estimation of time "held on" is entirely subjective, it could be argued that persons who experienced these phenomena as the result of a shock believe that they have been held on a long time, *i.e.* their appreciation of the passage of time is disturbed. Whilst this cannot be ruled out, the former hypothesis, that persons held on a longer time are more likely to experience such symptoms, is more feasible.

It is of interest therefore to determine why persons are held on for different periods. The problem can be conveniently studied in two parts:

TABLE 12

RELATIONSHIP OF PERIOD "HELD ON" AND CHEST SYMPTOMS

	Few Seconds	30-60 Seconds	Minutes	Unknown	Total
Chest symptoms	2	1	6	2	11
No chest symptoms	11	4	4	—	19
Total	13	5	10	2	30

TABLE 13

RELATIONSHIP OF PERIOD "HELD ON" AND HEART SYMPTOMS

	Few Seconds	30-60 Seconds	Minutes	Unknown	Total
Heart symptoms	1	1	2	2	6
No heart symptoms	12	4	8	—	24
Total	13	5	10	2	30

TABLE 14

RELATIONSHIP OF PERIOD "HELD ON" AND LOSS OF CONSCIOUSNESS

	Few Seconds	30-60 Seconds	Minutes	Unknown	Total
Consciousness lost	1	1	4	2	8
Consciousness not lost	12	4	6	—	22
Total	13	5	10	2	30

TABLE 15

METHOD OF RELEASE IN "HELD ON" CASES

Case No.	X Few Seconds	Case No.	Y 30-60 Seconds	Case No.	Z Minutes	Case No.	Unknown
10A	Foreman knocked him off	39	Thinks he fell off after 1 min.	4A	Unconscious, fell, pulled plug out	19	Pulled off by colleagues
11A	Moved to position and fell off			5	Release by mate	22	Fell off, jerked off
23A	Colleague switched off	46	Lost consciousness, came off (no chest symptoms)	16	Mate pulled him off		
32A	Threw drill away			27	Foreman pulled him off		
32B	Threw himself back, breaking contact	47	Suddenly became free (no chest or heart symptoms)	34A	A labourer kicked cable away		
34	Fell off, breaking control			37	Struggled free		
36	Kicked himself free	52	Ladder slipped, ? tetany	40A	Fell off (no chest or heart symptoms)		
48	Fuses blew	66A	Fell off (strangulation, white haze)	51A	Cable burned through		
48A	Fuses blew			67	Came off (chest tightness)		
65B	Jumped feet off ground (feet were part of circuit)			74	Came off (chest tightness)		
69A	Fuse blew						
76A	Kicked ladder away and fell off						
83A	Clonic movements of arms caused him to fall from perch, breaking contact						

(1) How are some victims released from an electrical circuit after being held on for only a few seconds, whilst others are held on for a period of minutes?

(2) How do some persons who have been held on for minutes (and who show a high incidence of impending asphyxia and of unconsciousness) get off, whilst others are known to remain in contact and die?

The second part of the problem might require clarification for it is often stated that a victim becomes unconscious and thereby lets go of the conductor. However, it is well established that isolated muscles can be electrically stimulated, and the intact organism is not needed for this response. It follows, therefore, that the onset of unconsciousness will not necessarily explain release from the circuit. There are three further possible explanations. (i) The onset of a severe degree of anoxia will be accompanied by relaxation of the muscles. This is unlikely because such a severe degree of anoxia would be likely to result in permanent damage to cells of the central nervous system and there was no evidence for this. (ii) Fatigue of the muscles might set in after prolonged strong stimulation. This might take place within a very few minutes (Merton, 1954). (iii) Some central action on the central nervous system may result in generalized loss of muscle tone (as in a faint). This would not affect the muscles under direct stimulation in the pathway of the current but as a result of this loss of tone the centre of gravity of the body might shift, causing the body weight to drag the victim off the conductor.

To study these problems the cases of "held on" shock were divided according to the length of time held on and the method of release noted. This is given briefly in Table 15.

To answer the first question (release of victims after a few seconds), attention is directed to column X. It will be seen that six persons were released by outside help or fortuitous circumstance (Cases 10A, 23A, 48, 48A, 69A, and 83A); of these it is impossible to say how many would have been held on for a longer period and might even have died. Of the remaining seven, all except Case 34 managed to do something to get themselves off the circuit. The answers given by Case 34 were not clear and it was not possible to be sure whether he fell intentionally to break the contact or as part of the accident. But apart from this it is apparent that those who were not released by some external agency were able to adopt some manoeuvre to get off the circuit. This could mean that they had the presence of mind to act before consciousness was impaired or, perhaps more likely, that they were able to adopt such a manoeuvre because they were subjected to a lesser current than the group who were held on longer.

The second question concerns the release of men

held on for a period estimated at over a minute. From column Z (Table 15) it is seen that five men were released by outside means (Cases 5, 16, 27, 34A, and 51A). Again it is impossible to state what would have been their fate if some external circumstances had not come to their aid. The remaining five merit individual study.

Case 4A.—This man became unconscious and fell, thereby pulling the electric plug out of a socket. The accident happened more than 30 years previously and it was not possible to establish whether he lost consciousness from asphyxia or otherwise.

Case 37.—The victim was held by the left arm and managed to struggle free with the right hand which was badly lacerated in the process. He was the only person in the group held on for minutes who freed himself.

Case 40A.—He was working at a hole in the road and fell off, fortunately not onto the conductor. He had no symptoms of impending asphyxia (although he had palpitation). He claims not to have lost consciousness. It is difficult to explain this unless one accepts that he had a brief faint.

Case 67 and 74.—Both of these men came off after chest and heart symptoms and were therefore being asphyxiated. Case 67 lost consciousness but 74 states that he did not.

This phenomenon of loss of consciousness resulting in release from a "held on" accident has been recorded also by Cambier (1953).

If it is to be accepted that the two victims who "came off" without losing consciousness (Cases 40A and 74), did so because of the onset of muscle fatigue, some residual muscle stiffness would be expected. This was not remarked upon by Case 40A, but Case 74 stated that he had stiffness in the abdominal muscles on the next day, as though he had had unaccustomed exercise. Obviously there is much individual variation because these, or any other possible mechanisms of release, do not operate in all cases; death by asphyxiation is well recognized by forensic pathologists as a cause of death from electric shock. In fact there is now another question: does muscle fatigue set in before death from asphyxia? And if so, why in some cases and not in others?

Physical Shock

There were three accidents associated with electricity but not causing any electro-pathological phenomena. During work on a live electric conductor there may be a sudden flashover, frequently associated with an explosion. This dramatic happening may not injure the workman and may not be associated with passage of current through the body. As these three accidents have common features they are considered together.

Case 65.—A jointer was using a wooden-handled hacksaw, held in the right hand, to cut through an 11,000 volt cable believed to be dead. He supported the cable with the toe of his left boot. There was a sudden explosion and a flash which vaporized the hacksaw blade. The jointer was quite definite that he did not receive a shock. The current had passed from the core of the cable and along the blade to the earthed armoured casing of the cable. He was thrown out of the pit by the explosion, temporarily blinded by the flash, and was trembling all over and feeling very cold. He was given first-aid treatment (warmth and warm drinks) by his mate and taken to the nearest hospital where he was detained for three hours and then resumed work.

There is little of medical significance in these cases of physical shock, except to note that such symptoms, when encountered after an electric shock, are not necessarily the result of the electric current but may equally well be due to the startling accident itself.

Artificial Respiration

The currently accepted emergency treatment in cases of electric shock is to apply artificial respiration. In this series only two cases reported that artificial respiration had been applied

Case 19.—A 21-year-old linesman's labourer, working outdoors in hob-nailed boots was standing on wet dewy soil, and grasped a copper wire in contact with a live conductor at 230 volts with the right hand. He could not let go and felt his right arm tightening and then his chest. He stated that he was losing consciousness when rescued and reported that his colleagues said he was going blue. They applied artificial respiration at once and he was taken to the medical unit of a nearby industrial firm (unfortunately the firm kept no record of this incident). He felt "shaky" for half an hour and rested for the remainder of the day although he felt all right. For a week afterwards he felt a stiffness in his neck and chest as though he had had unaccustomed exercise.

Comment.—This man received a "held on" shock from right arm to feet and was apparently being asphyxiated (as indicated by the blue appearance reported and the subsequent muscle stiffness) when he was rescued.

Case 58.—A 31-year-old fitter's mate was working in a sub-station when a three-phase flashover occurred on the 11,000 volt circuit. He received burns to the back of his hands and front of his face, neck, and right wrist (these necessitated a period in hospital of about three or four weeks). The man remembers little of what happened, having only "islands" of memory until about three days after the accident. Artificial respiration was administered at the time of the accident by his colleagues.

Comment.—In this case the victim received artificial respiration although he suffered only from flash burns and did not receive an electric shock. (This finding was confirmed by the safety officer's independent investigation.)

Treatment

At the present time the only first-aid treatment for apparent death due to electric shock is artificial respiration. Considerable attention is therefore given to training in the technique of artificial respiration by electricity supply undertakings and first-aid organizations. It is not surprising, therefore, that in the second case quoted above (Case 58) artificial respiration was applied, although it is unlikely that such measures would have been adopted had similar burns been caused by a non-electric source.

Whilst artificial respiration is the only treatment available to first-aiders at the present time, work is proceeding on a simple first-aid method of maintaining the circulation when the heart is not beating, by the application of cardiac massage to the closed chest (Kouwenhoven, Knickerbocker, Milnor, and Jude, 1960) and on a portable defibrillator (Kouwenhoven and Milnor, 1955, 1957; Kouwenhoven, 1960). These developments make it necessary to reassess the place of artificial respiration in electric shock and this study is in progress.

I am grateful to Professor R. E. Lane for his interest and advice during this study. It is a pleasure to thank the chairman of the Electricity Board, in whose area this work was done, the area secretary of the Electrical Trades Union for ready support, and the safety officers for their help and co-operation.

References

Bell, G. H., Davidson, J. N., and Scarborough, H. (1956). *Textbook of Physiology and Biochemistry*, 3rd ed., p. 467. Livingstone, Edinburgh.
Cambier, J. M. (1953). *Manifestations Nerveuses des Electrisations Accidentelles*. Foulon, Paris.
Dalziel, C. F. (1946). *Trans. Amer. Inst. elect. Engrs*, **65**, 579.
—— (1947). *Elect. Engng (N.Y.)*, **66**, 786.
——, Lagen, J. B., and Thurston, J. L. (1941). *Ibid*, **60**, 1073.
——, Ogden, E., and Abbott, C. E. (1943). *Ibid*, **62**, 745.
Emerson, S. J. (1961). Personal communication.
Ferris, L. P., King, B. G., Spence, P. W., and Williams, H. B. (1936). *Trans. Amer. Inst. elect. Engrs*, **55**, 498.
François, R. C. (1955). *Occup. Safety Hlth*, **5**, 5.
Hughes, J. P. W., and Corney, L. A. (1956). *Brit. J. industr. Med.*, **13**, 153.
Kouwenhoven, W. B. (1949). *Elect. Engng (N.Y.)*, **68**, 199.
—— (1960). Personal communication.
——, Hooker, D. R., and Langworthy, O. R. (1932). *Amer. J. Physiol.*, **100**, 344.
——, Knickerbocker, G. C., Milnor, W. R., and Jude, J. R. (1960). *Elect. Engng (N.Y.)*, **79**, 500.
——, and Milnor, W. R. (1955). *Ibid*, **74**, 218.
——, —— (1957). *Ibid*, **76**, 220.
Lewis, G. K. (1958). *J. Bone Jt Surg.*, **40A**, 27.
Merton, P. A. (1954). *J. Physiol. (Lond.)*, **123**, 553.
Minton, J. (1949). *Brit. med. J.*, **1**, 392.
Oliver, T. (1913). *Lancet*, **1**, 1575.
Rivers, E. C. (1894). *Arch. Ophthal. (N.Y.)*, **23**, 34.
Roy, D. (1897). *Amer. J. Ophthal.*, **14**, 353.
Tempest, M. N., and Atkins, J. B. (1958). *Brit. J. industr. Med.*, **15**, 147.

Brit. J. industr. Med., 1961, **18**, 270.

RHEUMATISM IN COTTON OPERATIVES

BY

J. S. LAWRENCE

From The Empire Rheumatism Council Field Unit, University of Manchester

(RECEIVED FOR PUBLICATION FEBRUARY 13, 1961)

Sixty-nine male and 103 female workers aged 45 and over in a cotton spinning mill in Bolton, Lancashire were examined clinically for rheumatic disease and had radiographs taken of the hands and feet and of the cervical, dorsal, and lumbar spine. To these were added the male and female cotton workers aged 45 and over from a random sample of the population of Leigh, giving a total of 117 males and 228 females. They were compared with a control group of 117 males and 228 females from random samples in Leigh and Wensleydale who had never worked in a cotton mill. These were matched by age and sex.

Rheumatic symptoms as a whole were less frequent in the cotton workers than in the controls and loss of work from rheumatic complaints was less frequent in the male cotton workers than in the male controls. Dorsal and chest pain were more common and caused more incapacity in both male and female cotton workers than in controls.

Heberden's nodes were present in 38% of male and 35% of female cotton workers compared with 12% and 22% respectively in the controls.

Radiological evidence of osteo-arthrosis was more frequent in the distal and proximal interphalangeal joints of the fingers and in the first carpo-metacarpal joints in the male cotton workers than in the controls. In the metacarpo-phalangeal joints of the fingers, the male cotton workers had much the same prevalence of osteo-arthrosis as the controls but it was more severe. The female cotton workers showed the same osteo-arthrosis joint pattern as the males and had similar prevalences in each joint, but did not differ substantially from the female controls.

There was no significant difference between the cotton workers and controls in respect of disk degeneration of the cervical spine, but in the dorsal and lumbar spine there was less disk degeneration in the cotton workers, the difference being greater in the females.

In a survey of osteo-arthrosis and disk degeneration in the town of Leigh in Lancashire, male cotton workers were found to have more osteo-arthrosis in the finger joints and in the first carpo-metacarpal joints of the hands than the remainder of the population. The numbers examined were small and the differences, for the most part, were not significant (Kellgren and Lawrence, 1958). It was therefore decided to supplement these findings by a survey of the workers in a cotton mill in the South Lancashire area.

Method

Before choosing a mill, data on age and sex distribution and sickness records were studied for 29 mills on which such information was available. Of these, Mill A in Bolton, Lancashire, was found to have sickness absence rates which fell nearest to the average for all mills in the group and was therefore chosen. Data on sickness absence are shown in Table 1. The greater absenteeism in cardroom workers and ring spinners may be due to the fact that they are females.

TABLE 1

AVERAGE SICKNESS ABSENTEEISM, IN DAYS PER EMPLOYEE, FOR 12 MONTHS ENDED MARCH 1957

	Cardroom	Mule Spinning	Ring Spinning	Doubling
All mills	7·5	5·6	8·9	5·0
Mill A	8·0	5·2	9·7	5·1

Following an initial approach to the management and to the trade union representatives at Mill A, all employees aged 45 and over were asked to come for examination to an *x*-ray van which was parked for the purpose in the mill yard. Employees who were

TABLE 2

COMPLETION RATE

Age (years)	Bolton				Leigh			
	Total in Sample	Not Available or Refused	Examined	Completion Rate (%)	Total in Sample	Not Available or Refused	Examined	Completion Rate (%)
Males								
45-54	31	3	28	90	169	18	151	89
55-64	37	5	32	86	102	9	93	91
65+	10	1	9	90	91	13	78	86
Total	78	9	69	88	362	40	322	89
Females								
45-54	74	14	60	81	171	19	152	89
55-64	40	2	38	95	138	26	112	81
65+	5	0	5	100	115	25	90	78
Total	119	16	103	86	424	70	354	83

TABLE 3

OCCUPATIONAL SUB-DIVISIONS IN LEIGH AND BOLTON COTTON WORKERS AGED 45 AND OVER

Occupation	Males					Females				
	Age (years)					Age (years)				
	Total	45-54	-64	-74	75+	Total	45-54	-64	-74	75+
Weavers	2	2	0	0	0	66	26	30	7	3
Spinners and doublers	81	35	30	14	2	69	45	23	1	0
Tenters and others	34	12	19	3	0	93	52	35	4	2
Total	117	49	49	17	2	228	123	88	12	5

not actually working with cotton, such as administrative and clerical staff, were excluded. A clinical examination was made of the musculo-skeletal system and routine radiographs were taken of the hands and feet and of the cervical, dorsal, and lumbar spine. Employees who were unwilling to come to the *x*-ray van, were examined in an office in the mill and had radiographs of hands, feet, and neck taken with a portable *x*-ray apparatus. Those who were absent on sick leave were either asked to come to the *x*-ray van, or had radiographs taken at their homes.

Altogether 69 males and 103 females were examined (Table 2) out of a total of 78 males and 119 females of appropriate age. This gave a completion rate of 88% in males and 86% in females. Only in the females aged 45-54 did the completion rate fall below 85%. Similar rates had been achieved in the random samples of the populations of Leigh and Wensleydale from which the control group was taken. The control group was chosen from those in the random sample who had never worked in a cotton mill and who had radiographs taken of hands, feet, cervical and lumbar, and (where available) dorsal spine. Radiographs of these sites were taken as a routine in the second half of the Leigh survey which took place in 1957. The 55-64 age group, however, had radiographs taken in a separate survey in 1954 and no radiographs of the dorsal spine were obtained. To fill this gap, a second sample aged 55-64 was examined in 1957 and radiographs of the dorsal spine only were taken. A matched sample of these has been used as controls in the data on dorsal disk degeneration. The predominant occupations of the controls were coal-mining in males and domestic in females.

For the purposes of this study, all those aged 45 and over in the Leigh random sample whose predominant occupation had been in the cotton mills were added to the Mill A cotton workers. This gave a total of 117 males and 228 females (Table 3). The largest proportion was in the 45-54 age group in the females but was equally distributed between this and the 55-64 age group in males. The cotton workers were divided into three groups according to their occupation in the mills. Spinning was the most frequent occupation in males but in females the occupations were fairly evenly distributed between weavers, spinners, and tenters (machine tenders), the last being the largest. This was a miscellaneous group comprising mainly cardroom workers.

Clinical Features

Altogether 85 (73%) of the 117 male cotton workers gave a history of rheumatic symptoms, com-

Brit. J. industr. Med., 1961, **18**, 270.

RHEUMATISM IN COTTON OPERATIVES

BY

J. S. LAWRENCE

From The Empire Rheumatism Council Field Unit, University of Manchester

(RECEIVED FOR PUBLICATION FEBRUARY 13, 1961)

Sixty-nine male and 103 female workers aged 45 and over in a cotton spinning mill in Bolton, Lancashire were examined clinically for rheumatic disease and had radiographs taken of the hands and feet and of the cervical, dorsal, and lumbar spine. To these were added the male and female cotton workers aged 45 and over from a random sample of the population of Leigh, giving a total of 117 males and 228 females. They were compared with a control group of 117 males and 228 females from random samples in Leigh and Wensleydale who had never worked in a cotton mill. These were matched by age and sex.

Rheumatic symptoms as a whole were less frequent in the cotton workers than in the controls and loss of work from rheumatic complaints was less frequent in the male cotton workers than in the male controls. Dorsal and chest pain were more common and caused more incapacity in both male and female cotton workers than in controls.

Heberden's nodes were present in 38% of male and 35% of female cotton workers compared with 12% and 22% respectively in the controls.

Radiological evidence of osteo-arthrosis was more frequent in the distal and proximal interphalangeal joints of the fingers and in the first carpo-metacarpal joints in the male cotton workers than in the controls. In the metacarpo-phalangeal joints of the fingers, the male cotton workers had much the same prevalence of osteo-arthrosis as the controls but it was more severe. The female cotton workers showed the same osteo-arthrosis joint pattern as the males and had similar prevalences in each joint, but did not differ substantially from the female controls.

There was no significant difference between the cotton workers and controls in respect of disk degeneration of the cervical spine, but in the dorsal and lumbar spine there was less disk degeneration in the cotton workers, the difference being greater in the females.

In a survey of osteo-arthrosis and disk degeneration in the town of Leigh in Lancashire, male cotton workers were found to have more osteo-arthrosis in the finger joints and in the first carpo-metacarpal joints of the hands than the remainder of the population. The numbers examined were small and the differences, for the most part, were not significant (Kellgren and Lawrence, 1958). It was therefore decided to supplement these findings by a survey of the workers in a cotton mill in the South Lancashire area.

Method

Before choosing a mill, data on age and sex distribution and sickness records were studied for 29 mills on which such information was available. Of these, Mill A in Bolton, Lancashire, was found to have sickness absence rates which fell nearest to the average for all mills in the group and was therefore chosen. Data on sickness absence are shown in Table 1. The greater absenteeism in cardroom workers and ring spinners may be due to the fact that they are females.

TABLE 1

AVERAGE SICKNESS ABSENTEEISM, IN DAYS PER EMPLOYEE, FOR 12 MONTHS ENDED MARCH 1957

	Cardroom	Mule Spinning	Ring Spinning	Doubling
All mills	7·5	5·6	8·9	5·0
Mill A	8·0	5·2	9·7	5·1

Following an initial approach to the management and to the trade union representatives at Mill A, all employees aged 45 and over were asked to come for examination to an *x*-ray van which was parked for the purpose in the mill yard. Employees who were

TABLE 2

COMPLETION RATE

Age (years)	Bolton				Leigh			
	Total in Sample	Not Available or Refused	Examined	Completion Rate (%)	Total in Sample	Not Available or Refused	Examined	Completion Rate (%)
Males								
45-54	31	3	28	90	169	18	151	89
55-64	37	5	32	86	102	9	93	91
65+	10	1	9	90	91	13	78	86
Total	78	9	69	88	362	40	322	89
Females								
45-54	74	14	60	81	171	19	152	89
55-64	40	2	38	95	138	26	112	81
65+	5	0	5	100	115	25	90	78
Total	119	16	103	86	424	70	354	83

TABLE 3

OCCUPATIONAL SUB-DIVISIONS IN LEIGH AND BOLTON COTTON WORKERS AGED 45 AND OVER

Occupation	Males					Females				
	Age (years)					Age (years)				
	Total	45-54	–64	–74	75+	Total	45-54	–64	–74	75+
Weavers	2	2	0	0	0	66	26	30	7	3
Spinners and doublers	81	35	30	14	2	69	45	23	1	0
Tenters and others	34	12	19	3	0	93	52	35	4	2
Total	117	49	49	17	2	228	123	88	12	5

not actually working with cotton, such as administrative and clerical staff, were excluded. A clinical examination was made of the musculo-skeletal system and routine radiographs were taken of the hands and feet and of the cervical, dorsal, and lumbar spine. Employees who were unwilling to come to the *x*-ray van, were examined in an office in the mill and had radiographs of hands, feet, and neck taken with a portable *x*-ray apparatus. Those who were absent on sick leave were either asked to come to the *x*-ray van, or had radiographs taken at their homes.

Altogether 69 males and 103 females were examined (Table 2) out of a total of 78 males and 119 females of appropriate age. This gave a completion rate of 88% in males and 86% in females. Only in the females aged 45-54 did the completion rate fall below 85%. Similar rates had been achieved in the random samples of the populations of Leigh and Wensleydale from which the control group was taken. The control group was chosen from those in the random sample who had never worked in a cotton mill and who had radiographs taken of hands, feet, cervical and lumbar, and (where available) dorsal spine. Radiographs of these sites were taken as a routine in the second half of the Leigh survey which took place in 1957. The 55-64 age group, however, had radiographs taken in a separate survey in 1954 and no radiographs of the dorsal spine were obtained. To fill this gap, a second sample aged 55-64 was examined in 1957 and radiographs of the dorsal spine only were taken. A matched sample of these has been used as controls in the data on dorsal disk degeneration. The predominant occupations of the controls were coal-mining in males and domestic in females.

For the purposes of this study, all those aged 45 and over in the Leigh random sample whose predominant occupation had been in the cotton mills were added to the Mill A cotton workers. This gave a total of 117 males and 228 females (Table 3). The largest proportion was in the 45-54 age group in the females but was equally distributed between this and the 55-64 age group in males. The cotton workers were divided into three groups according to their occupation in the mills. Spinning was the most frequent occupation in males but in females the occupations were fairly evenly distributed between weavers, spinners, and tenters (machine tenders), the last being the largest. This was a miscellaneous group comprising mainly cardroom workers.

Clinical Features

Altogether 85 (73%) of the 117 male cotton workers gave a history of rheumatic symptoms, com-

pared with 104 (89%) of the controls (Table 4). Loss of work from rheumatism had occurred in 39 (34%) of the male cotton workers and 57 (49%) of the controls. Only in the dorsal region was there a substantially greater prevalence of symptoms in the male cotton workers. This was associated with either no loss of work or loss of less than three months in all instances in the cotton workers.

In the females similarly (Table 5) there were in general fewer rheumatic complaints in the cotton workers (74% compared with 85% in the controls). There was however, no significant difference in incapacity; 32% of cotton workers lost work from rheumatism, compared with 28% of the controls. The dorsal spine was the only area in which symptoms were more frequent in female cotton workers and this area and the cervical spine were mainly responsible for the slightly greater incapacity in these workers.

Altogether 63 of the male controls had worked in coal-mines. When these were excluded, the symptom rate in the controls was still slightly greater than in the cotton workers.

Physical Signs.—The only striking feature on clinical examination of the cotton workers was the frequency of Heberden's nodes which were present in 38% of males and 35% of females, and in 12% and 22% of the controls.

Radiological Findings

The radiographs of the cotton workers and controls were mixed together before reading so that the observer was unaware of their source. They were

TABLE 4

RHEUMATIC COMPLAINTS AND LOSS OF WORK IN 117 MALE COTTON WORKERS AND CONTROLS

Site of Pain	Cotton Workers			Controls		
	Number With Pain	Number Off Work		Number With Pain	Number Off Work	
		1-11 Weeks	3 Months		1-11 Weeks	3 Months+
Distal interphalangeal joints	7	1	2	8	0	4
Proximal interphalangeal joints	14	2	2	12	0	4
Metacarpo-phalangeal joints	9	1	2	7	0	4
Carpo-metacarpal joints	6	1	2	4	0	3
Wrists	6	1	2	11	1	4
Cervical spine	34	5	2	48	12	4
Dorsal spine	19	6	0	8	0	1
Lumbar spine	43	19	5	59	16	11
Hips	4	0	2	12	2	3
Sacro-iliac joints	3	0	2	5	2	1
Knees	28	4	4	41	5	10
Tarsi	15	3	3	12	1	4
Lateral metatarso-phalangeal joints	6	1	2	8	0	4
First metatarso-phalangeal joints	8	1	2	12	1	4
No. of workers and controls affected	85 (73%)	30 (26%)	9 (8%)	104 (89%)	34 (29%)	23 (20%)

TABLE 5

RHEUMATIC COMPLAINTS AND LOSS OF WORK IN 228 FEMALE COTTON WORKERS AND CONTROLS

Site of Pain	Cotton Workers			Controls		
	Number With Pain	Number Off Work		Number With Pain	Number Off Work	
		1-11 Weeks	3 Months		1-11 Weeks	3 Months +
Distal interphalangeal joints	28	4	5	44	6	11
Proximal interphalangeal joints	35	4	6	55	6	12
Metacarpo-phalangeal joints	32	4	7	53	6	12
Carpo-metacarpal joints	32	2	9	39	6	11
Wrists	19	6	4	40	6	10
Cervical spine	88	17	7	103	12	7
Dorsal spine	27	8	3	23	2	2
Lumbar spine	81	25	9	103	28	6
Hips	8	0	3	20	2	7
Sacro-iliac joints	7	0	3	11	3	5
Knees	56	8	8	93	15	23
Tarsi	39	11	10	57	9	12
Lateral metatarso-phalangeal joints	19	0	5	33	5	10
First metatarso-phalangeal joints	26	3	7	35	5	9
Pain at all sites	177 (74%)	51 (21%)	27 (11%)	195 (85%)	40 (18%)	21(10%)

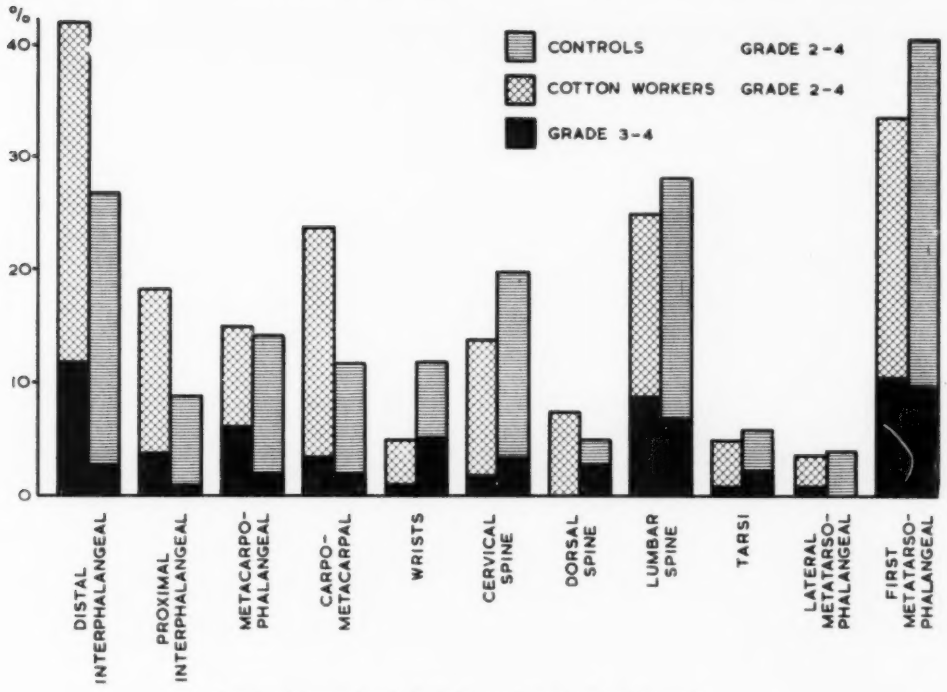


FIG. 1.—Osteo-arthrosis in male cotton workers and controls.

graded for osteo-arthrosis and rheumatoid arthritis and in the spine films for disk degeneration. Each diagnosis was graded 0-4 for each type of joint, using standard films (Kellgren and Lawrence, 1957; Lawrence and Bremner, 1961).

Osteo-arthrosis.—Radiological evidence of osteo-arthrosis was found to be more common in male cotton workers than controls in four types of joint and less common in seven (Fig. 1). The joints which were more affected in the cotton workers were all in the hands and included the distal and proximal interphalangeal, the metacarpo-phalangeal and first carpo-metacarpal joints. There was no difference in the frequency and severity of these changes between the right and left hands. Only in the wrists was there appreciably more osteo-arthrosis in the controls ($\chi^2 = 3{\cdot}2$, $p = 0{\cdot}08$). The difference between grade

TABLE 6

OSTEO-ARTHROSIS JOINT PATTERN IN FEMALE COTTON WORKERS AND CONTROLS

	Cotton Workers								Controls							
	Number Radio-graphed	Grade					2-4 (%)	3-4 (%)	Number Radio-graphed	Grade					2-4 (%)	3-4 (%)
		0	1	2	3	4				0	1	2	3	4		
Distal interphalangeal joints	225	72	49	79	17	8	46	11	226	108	30	66	15	7	39	10
Proximal interphalangeal joints	225	145	30	38	10	2	22	5	226	159	28	28	7	4	17	5
Metacarpo-phalangeal joints	225	155	46	21	2	1	11	1	226	163	34	24	4	1	13	2
Carpo-metacarpal joints	225	137	37	31	14	6	23	9	226	159	29	25	11	2	17	6
Wrists	225	195	18	8	4	0	5	2	225	205	12	8	0	0	6	0
Cervical spine	222	153	45	20	3	1	11	2	223	139	51	26	6	1	15	3
Dorsal spine	128	88	31	9	0	0	7	0	41*	23	11	7	0	0	17	0
Lumbar spine	200	83	77	32	7	1	20	4	214	84	93	30	3	4	17	3
Tarsi	222	189	23	3	7	0	5	3	224	198	14	8	4	0	5	2
Lateral metatarso-phalangeal joints	222	193	17	11	1	0	5	0·4	225	203	14	5	1	2	4	1
First metatarso-phalangeal joints	222	80	51	68	20	3	41	10	224	111	41	54	14	4	32	8

*The control group of dorsal spine radiographs in the 55-64 age group were drawn from a special sample radiographed in 1957.

FIG. 2.—Radiograph of hand of male cotton operative.

3-4 osteo-arthrosis in the distal interphalangeal joints in cotton workers and that in controls was highly significant ($\chi^2 = 7{\cdot}2$, $p = <0{\cdot}01$).

In females the pattern of osteo-arthrosis and the general prevalence in cotton workers were very similar to those in the controls (Table 6). This was largely a result of the high prevalence in the female controls who had as much osteo-arthrosis as the male cotton workers. Such differences as occurred between the female cotton workers and controls were similar to those between male cotton workers and controls but were in no instance significant (Table 7).

The radiographic changes in the hand are illustrated in Fig. 2.

Disk Degeneration.—In males the prevalence and severity of cervical disk degeneration were identical

TABLE 7

OSTEO-ARTHROSIS JOINT PATTERN IN MALE COTTON WORKERS AND CONTROLS

	Cotton Workers								Controls							
	Number Radio-graphed	Grade					2-4 (%)	3-4 (%)	Number Radio-graphed	Grade					2-4 (%)	3-4 (%)
		0	1	2	3	4				0	1	2	3	4		
Distal interphalangeal joints	115	40	27	34	11	3	42	12	117	59	26	29	2	1	27	3
Proximal interphalangeal joints	115	78	14	19	3	1	20	3	117	102	4	10	1	0	9	1
Metacarpo-phalangeal joints	115	83	15	10	5	2	15	6	117	88	13	14	1	1	14	2
Carpo-metacarpal joints	115	70	16	25	3	1	25	3	117	81	22	11	3	0	12	3
Wrists	115	97	12	5	1	0	5	1	116	85	17	8	6	0	12	5
Cervical spine	113	67	30	14	2	0	14	2	115	54	38	19	2	2	20	3
Dorsal spine	72	52	16	4	0	0	8	0	60*	37	20	1	2	0	5	3
Lumbar spine	103	40	36	18	9	0	26	9	102	35	37	23	7	0	29	7
Tarsi	115	94	15	5	1	0	5	1	116	100	9	4	2	1	5	3
Lateral metatarso-phalangeal joints	115	106	4	4	1	0	4	1	116	108	3	5	0	0	4	0
First metatarso-phalangeal joints	115	58	18	26	13	0	34	11	116	48	20	36	10	2	41	10

*The control group of dorsal spine radiographs in the 55-64 age group were drawn from a special sample radiographed in 1957.

TABLE 8

DISK DEGENERATION IN COTTON WORKERS AND CONTROLS

	Cotton Workers								Controls							
	Number Radiographed	Grade					Four or More Disks Affected	Grade		Number Radiographed	Grade					Four or More Disks Affected	Grade	
		0	1	2	3	4		2-4 (%)	3-4 (%)		0	1	2	3	4		2-4 (%)	3-4 (%)
Males																		
Cervical spine	113	29	5	31	43	5	12	70	42	115	19	16	36	36	8	12	70	38
Dorsal spine	72	16	3	40	11	2	39	74	18	60	4	4	35	15	2	33	86	28
Lumbar spine	103	21	15	56	9	2	15	65	10	107	24	7	54	21	1	32	71	21
Females																		
Cervical spine	222	86	17	62	52	5	8	76	26	223	68	25	66	57	7	18	61	30
Dorsal spine	128	33	5	71	18	1	60	70	15	41*	2	2	21	15	1	27	90	39
Lumbar spine	200	87	35	67	10	1	17	39	6	214	70	46	69	22	7	15	46	14

*See footnote to Table 6.

in cotton workers and controls, but dorsal disk degeneration was slightly less frequent in the cotton workers, of whom 74% had definite evidence of this disease in one or more disks, compared with 86% in the controls (Table 8). Lumbar disk degeneration was also less frequent in the male cotton workers than in the controls, particularly the more severe forms.

The female cotton workers had slightly more cervical disk degeneration than the controls but of less severity, and on balance it appears there is no occupational influence. Both dorsal and lumbar disk degeneration, however, were appreciably less in the female cotton workers than in the controls. The number of female controls in whom a dorsal radiograph was available was small, as routine radiographs of this region were taken in only a third of the respondents. It is, however, clear that the greater loss of working time from dorsal symptoms in cotton workers cannot be attributed to disk degeneration, nor was any other objective cause found.

Occupational Sub-divisions in Cotton Workers

In Table 9 the cotton workers have been divided into three groups, weavers, spinners, and " others ", and the prevalence of osteo-arthrosis in the distal and proximal interphalangeal joints and the first carpo-metacarpal joints in these three groups is compared. There were insufficient male weavers for any conclusions to be reached, but the spinners and " others " may be compared. Both spinners and " others " had more osteo-arthrosis in the first carpo-metacarpal joints than the controls (*cf.* Table 7), but the spinners had the greatest prevalence in these joints and also in the distal interphalangeals. In the proximal interphalangeals there was little difference. In female cotton workers there was more disease in all joints in the weavers but this group had more old people. When corrected for age, this difference largely disappeared. Between the spinners and " others ", there was also little difference, though the proximal interphalangeal joints were less affected in the spinners than in the " others ".

It appears that all the main types of occupation in the cotton mill are associated with an increased prevalence of osteo-arthrosis in these three joints.

Rheumatoid arthritis was encountered in 3% of male and 7% of female cotton workers and spondylitis and gout each in 1% of male cotton workers.

TABLE 9

OSTEO-ARTHROSIS OF THE DISTAL INTERPHALANGEAL AND PROXIMAL INTERPHALANGEAL AND FIRST CARPO-METACARPAL JOINTS IN SPINNERS, WEAVERS, AND OTHERS

Occupation	Number Radiographed	Distal Interphalangeal Joints						Proximal Interphalangeal Joints						First Carpo-metacarpal Joints					
		Grade						Grade						Grade					
		0	1	2	3	4	2-4 (%)	0	1	2	3	4	2-4 (%)	0	1	2	3	4	2-4 (%)
Males																			
Weavers	2	2	—	—	—	—	—	2	—	—	—	—	—	2	—	—	—	—	—
Spinners	80	21	22	26	8	3	46	53	10	14	2	1	21	44	13	20	3	0	29
Others	33	17	5	8	3	0	33	23	4	5	1	0	18	24	3	5	0	1	18
Females																			
Weavers	66	14	14	29	7	2	57	29	16	15	6	0	32	29	14	13	6	4	35
Spinners and doublers	69	24	17	22	4	2	41	56	5	7	0	1	12	51	9	7	2	0	26
Others	90	34	18	28	6	4	42	60	9	16	4	1	22	57	14	11	7	1	21

These prevalences are not significantly different from those in the general population. There is thus no evidence of an occupational factor in these diseases so far as cotton workers are concerned.

Discussion

No comparison of workers in a particular occupation with those in all remaining occupations in the area can be said to give a complete picture of the effect of occupation on disease, since occupational factors may well play a part in the control group. In the town of Leigh, from which the control group was mainly drawn, a third of the male workers were coal-miners. It is known that mining has a considerable effect on the incidence of osteo-arthrosis in many joints and also on disk degeneration (Kellgren and Lawrence, 1952; Lawrence, 1955). This accounts for the higher prevalence of osteo-arthrosis in the wrists in the controls and tends to hide increased prevalences in other joints in the cotton workers. It would, however, be unreasonable to remove only miners from the control group since almost all occupations probably influence osteo-arthrosis to some extent.

The picture is further complicated by the influence of heredity which affects both the frequency and pattern of osteo-arthrosis. In females, Heberden's nodes together with osteo-arthrosis of the inter-phalangeal, metacarpo-phalangeal, and first carpo-metacarpal joints are greatly influenced by genetic factors (Lawrence, 1961). It is not surprising therefore, that the differences in joint pattern in females between cotton workers and controls are relatively small and insignificant.

The lower general symptom rate in cotton workers is of interest particularly as it is associated in the males with reduced loss of work from rheumatic troubles. This does not appear to be due to a reduced prevalence or severity of rheumatic disease in the cotton workers who indeed have more severe disease in certain joints. A dissociation between the radiographic changes of degenerative joint disease and symptoms has also been noted in coal-miners, those working in wet seams having more symptoms and incapacity than those in dry seams, despite identical severity of radiographic changes (Lawrence, 1955). This was attributed to a reduced pain threshold from cooling of tissues at the sites of referred pain. In cotton mills the atmosphere is humid but very warm, and this may well account for the dissociation found in these workers. It is of interest that in the weavers, who work in relatively cool conditions, symptoms were more frequent, 88% having rheumatic complaints compared with 74% in the spinners and cardroom workers and 85% in the controls. This was not associated with a reduction of loss of work from rheumatic complaints.

The increased prevalence of osteo-arthrosis observed in the finger and thumb joints in these cotton workers did not appear to be due to overt trauma since few gave a history of injury. Two factors may play a part. The skilled cotton operative is using his fingers continuously and rapidly, and the joints may be subject to continuous minor traumata, for example, knocking against frames and bobbins. Secondly he is working in a warm atmosphere with a consequent raising of his pain threshold so that the effects of slight trauma are less likely to be noticed. The combination of these two factors may well be responsible for the severe changes sometimes encountered.

In view of the relatively benign nature of the disease found in cotton operatives, preventive measures are not indicated.

I wish to express my thanks to the manager and workers in the Mill for their kind co-operation. I am indebted to Messrs. Fine Spinners and Doublers of Manchester, who not only gave permission to carry out this survey, but also supplied data on sickness absence in their mills.

I am also indebted to Professor R. E. Lane and Dr. W. R. Lee of the Department of Occupational Health, Manchester University, for their advice.

References

Kellgren, J. H., and Lawrence, J. S. (1952). *Brit. J. industr. Med.*, **9**, 197.
——, —— (1957). *Ann. rheum. Dis.*, **16**, 494.
——, —— (1958). *Ibid*, **17**, 388.
Lawrence, J. S. (1955). *Brit. J. industr. Med.*, **12**, 249.
—— (1961). *Ann. rheum. Dis.*, **20**, 11.
——, and Bremner, J. (1961). *Ibid.* (in the press).

Brit. J. industr. Med., 1961, **18**, 277.

FURTHER STUDIES ON THE TOXICITY OF SOME TETRA AND TRIALKYL LEAD COMPOUNDS

BY

JILL E. CREMER and S. CALLAWAY

From The Toxicology Research Unit, M.R.C. Laboratories, Carshalton, Surrey, and The War Office Chemical Defence Experimental Establishment, Porton Down, Salisbury, Wilts

(RECEIVED FOR PUBLICATION MAY 18, 1961)

The toxicity of tetra and trimethyl and propyl lead compounds has been studied after their administration to rats and rabbits. The toxicity to rats of tetramethyl lead has been compared with tetraethyl lead when given by inhalation.

Both tetramethyl and tetrapropyl lead were found to be considerably less toxic than trimethyl and tripropyl lead. There was evidence of a slow rate of conversion of the tetra to the trialkyl lead forms in rats *in vivo*.

Although there was a distinct difference between the signs of poisoning seen after giving the methyl or the propyl lead compounds the primary site of action for both groups appeared to be the central nervous system.

Some biochemical studies using slices of rat brain cortex showed that trimethyl and tripropyl lead inhibited the oxidation of glucose whereas tetramethyl and tetrapropyl lead were a hundred times less active in this respect.

In a previous paper (Cremer, 1959) some biochemical studies on the toxicity of tetraethyl lead were described. The present paper reports similar work with tetra and trimethyl lead and tetra and tripropyl lead.

Because of its toxic properties towards man the importance of care in handling tetraethyl lead is well known. Recent developments in the petroleum industry have shown that tetramethyl lead is likely to become of importance as an improved anti-knock additive. Some preliminary studies on its toxicity in laboratory animals have therefore been undertaken together with studies on closely related compounds.

MATERIALS AND METHODS

Pure samples of the lead compounds were supplied by Mr. G. F. Harrison, of The Associated Ethyl Company Ltd., Ellesmere Port, Cheshire and by Professor R. A. Kehoe, The Kettering Laboratory, Department of Preventive Medicine and Industrial Health, College of Medicine, University of Cincinnati, Cincinnati, Ohio.

Adult albino rats of 175 to 230 g. and rabbits of 2·5 to 3·0 kg. body weight were used.

Administration of Lead Compounds in Animals

Intravenous injections of tetramethyl lead were given to rats (tail vein) and rabbits (ear vein) as a solution in ethanol. Each rat received not more than 0·05 ml. and each rabbit not more than 0·2 ml. of the solution. Oral administration of the alkyl lead compounds to rats was by stomach tube. Tetramethyl and tetrapropyl lead were dissolved in arachis oil. Tripropyl lead chloride was dissolved in one part dimethyl formamide and two parts arachis oil.

Trimethyl lead chloride was given to rats and rabbits by intraperitoneal injection as a solution in 0·9% NaCl. Tripropyl lead chloride was given to rats by intraperitoneal injection as a solution in glycerol formol.

The inhalation exposures were carried out in an all-glass chamber. Groups of four rats were exposed simultaneously and the apparatus was designed so that only the heads of the rats were in contact with the vapour. Pure solutions of tetramethyl and tetraethyl lead were injected into the chamber by a mechanically driven syringe. The air-flow was maintained at 50 litres per minute. Samples of air were taken at intervals during the 60-minute exposure period into Neal bubblers at approximately 1 litre per minute through accurately calibrated critical orifices. The bubblers contained 5 ml. of 100-200 petroleum ether.

Estimation of Trialkyl Lead Compounds and Inorganic Lead

The trialkyl lead compounds were estimated by the dithizone procedure (Cremer, 1959). Neither tetra-

methyl nor tetrapropyl lead formed a coloured complex with dithizone.

The petroleum ether containing tetra-alkyl lead compounds trapped in the bubblers was washed into 100 ml. flasks, using 1 or 2 ml. of petroleum ether, and 5 ml. concentrated hydrochloric acid were added. The mixture was shaken for five minutes and then placed in a boiling water bath for 30 minutes. The aqueous layer was made up to 100 ml. with distilled water and the remaining petroleum ether was sucked off. The resulting solution of lead chloride was estimated polarographically. This method was modified from Milner (1957).

The lethal doses and their 95% confidence limits were calculated by the method of Weil (1952).

The procedure for studying the metabolism of glucose by slices of rat brain cortex was exactly as described previously (Cremer, 1959). Details are given in the legend to Table 4.

RESULTS

Action of Organo-lead Compounds *in vivo*

Toxicity to Rats.—Summaries of the response of rats to the different lead compounds are given in Tables 1 and 2. Within a few minutes of receiving intraperitoneal injections of trimethyl lead chloride the rats were lying stretched out with paresis of the hind legs. The ears and tails were very pale. These signs largely disappeared 40 minutes later, giving way to general signs of toxicity; the animals sat quietly with staring coats. Rats which did not die within the first two hours but which received lethal doses became hyperexcitable and irritable with sparring behaviour. Generalized body tremors appeared which became increasingly severe with occasional convulsions. Some animals exhibited marked tremors for as long as 24 hours before death.

Tetramethyl lead was appreciably less toxic to rats than trimethyl lead. Because of the toxicity of the solvent the maximum dose given by intravenous injection was about 34 mg./kg. No signs of poisoning appeared in rats receiving this dose. Higher doses of tetramethyl lead which were given orally, produced signs of poisoning identical with those produced by trimethyl lead chloride.

After exposure by inhalation to the highest concentration of tetramethyl lead (9·84 mg./litre for 60 minutes) no obvious signs of intoxication were seen beyond a mild hyperexcitability. The death of one rat with bronchopneumonia was not thought to be attributable to tetramethyl lead. The development of signs of poisoning after inhalation of tetraethyl lead followed a pattern identical with that described after giving tetraethyl lead intravenously (Cremer, 1959). The hyperexcitable state and continuous generalized body tremors seen in these animals were the same as those seen in rats poisoned with trimethyl lead chloride or high doses of tetramethyl lead.

Rats given tripropyl lead chloride responded differently from those given trimethyl or triethyl lead chloride. After an intraperitoneal dose of 20 mg./kg. the rats showed almost immediate signs of uneasiness and at four hours two out of a group of four rats were weak with an unsteady gait. One of these two rats died overnight and the other became steadily weaker especially in the hind legs, and died quietly 28 hours after the injection. The remaining two rats became very irritable and developed a characteristic gait with stiff back legs, a humped back and the tail held high. The rats remained in this condition for six days after which they regained a normal posture and movement. After giving higher doses of tripropyl lead orally a similar response was seen. Definite weakness of the hind limbs was a constant feature and rats which became weak had difficulty in remaining in an upright position when they closed their eyes. In rats given up to 40 mg./kg. of tetrapropyl lead intravenously, no signs of poisoning were seen. Four rats given a high oral dose of tetrapropyl lead (395 mg./kg.) developed signs of poisoning identical with those seen after giving tripropyl lead. Tremors and convulsions were not seen after giving the propyl lead compounds.

Toxicity to Rabbits.—Two adult rabbits were given trimethyl lead chloride by intraperitoneal injection. The doses were 7·5 and 15 mg./kg. Both rabbits showed an immediate response of lying stretched out with a wriggling body movement. The rabbit which received the higher dose died 12 hours after injection. During the first week the remaining rabbit appeared to be hyperaesthetic when handled around the head but this reaction became less marked during the following two weeks and the animal seemed otherwise normal.

Two adult rabbits were injected intravenously with tetramethyl lead at 20 and 40 mg./kg. No signs of poisoning could be discerned during a three-week period following the injections. A rabbit injected intravenously with tetraethyl lead (31 mg./kg.) showed immediate signs of distress with very rapid breathing and stretching out in its cage. It died unobserved approximately 16 hours after injection.

Distribution of Organo-lead Compounds in Rats

The dithizone method used for estimating trialkyl lead compounds in tissues specifically measures the complete trialkyl lead ions without interference from tetra or dialkyl lead or inorganic lead ions.

The distribution of trimethyl lead and tripropyl lead in certain tissues of rats killed four hours and

TABLE 1

TOXICITY OF ORGANO-LEAD COMPOUNDS TO RATS

Compound	No. of Animals	Dose (mg./kg.)	Route	Reactions	Death	Time of Death After Injection (days)	LD_{50} (mg./kg.)
Trimethyl lead chloride	2	100	I.P.		2	2 hours	
	2	50	I.P.	Generalized body tremors after 24 hours	2	2	
	2	38·4	I.P.	Generalized body tremors	2	1 and 4	25·5 (approximate value)
	2	29·5	I.P.	Hyperexcitable with altered gait	2	7 and 9	
	4	23	I.P.	Excitable during first week with a slight alteration of gait and small loss of body weight; surviving rats returned to normal	1	28 hours	
	4	16	I.P.	No signs of poisoning	0		
Tetramethyl lead	4	26	I.V.	No signs of poisoning	0		
	4	34·3	I.V.	No signs of poisoning	0		
	4	62·5	Orally	Loss of weight during first week, thereafter regained weight	0		
	4	125	Orally	Tremors developed after three days; surviving rat became ill with altered gait, but gradually returned to normal behaviour	3	5-8	109·3
	4	250	Orally	Very excitable behaviour, sparring, tremors, and convulsions	4	4	(77·43-154·5)
	4	500	Orally	Tremors developed after 24 hours	4	2 and 3	
Tripropyl lead chloride	4	5	I.P.	No signs of poisoning	0		
	4	10	I.P.	Lost weight during first few days, thereafter surviving rats regained weight; all rats showed humped back gait but three gradually returned to normal	1	2	
	4	20	I.P.	One rat became weak within three hours after injection and remained so until death; the two surviving rats had altered gait but returned gradually to normal; some initial weight loss	2	1 and 2	
	4	40	Orally	After two days all four rats looked ill and had altered gait; two rats became weaker; remaining two rats gradually improved	2	4 and 7	26·67 (approximate value)
	4	80	Orally	Between 24 and 72 hours all rats became very weak with unsteady gait	4	3 to 6	
Tetrapropyl lead	4	30·8	I.V.	No signs of poisoning	0		
	4	40	I.V.	No signs of poisoning	0		
	4	395	Orally	After 48 hours the rats looked ill with a weakness of the hind legs; weakness became more generalized until death	4	3 to 4	

TABLE 2

A COMPARISON OF THE TOXICITY TO RATS OF TETRAMETHYL AND TETRAETHYL LEAD GIVEN BY INHALATION

Compound	Nominal Concentration (mg./litre)	Analysis Mean Range (mg./litre)	Reactions	Deaths	Approximate Dose (mg./kg.)*
Tetraethyl lead	4·84	3·87 (3·3-4·4)	Eight rats died in under six hours and remaining rats died in under 12 hours; intensely cyanotic at death	12/12	232
	2·77	2·64 (2·6-2·9)	All died in less than 24 hours	20/20	158
	1·58	1·50 (1·32-1·69)	Three died in 24 hours; remaining rats died in under 72 hours	16/16	90
	0·77	0·66 (0·57-0·74)	All rats showed excitable sparring behaviour; one rat died after two days; two rats showed continuous tremors leading to death in 14 days	3/16	39·6
	0·37	0·34 (0·32-0·39)	All affected with mild signs of poisoning with slow recovery	0/20	20·4
	0·19	0·17 (0·15-0·18)	All affected with mild signs of poisoning with slow recovery	0/16	10·2
	0·095	0·08	Very mild signs of poisoning from which recovery was made	0/16	4·8
Tetramethyl lead	9·84	8·87 (8·42-9·26)	One rat died after 16 days; macroscopical examination suggested a bronchopneumonia of unknown origin; a mild hyperexcitability seen in all rats but no other obvious signs of intoxication	1/16	533
	4·92	4·48 (4·37-4·78)	No obvious signs of intoxication other than a mild hyperexcitability	0/12	269
	2·41	2·18 (2·07-2·29)	No obvious signs of poisoning	0/16	131

Assuming a probit slope of about 8 the LC_{50} for tetraethyl lead is approximately 0·85 mg./litre (based on the analytical concentration of tetraethyl lead). For tetramethyl lead the LC_{50} is higher than 8·87 mg./litre.

*The approximate dose of the tetra-alkyl lead compounds has been calculated as mg./kg. body weight based on an average air intake for a rat of 100 ml./100 g. body weight/minute.

TABLE 3

DISTRIBUTION OF TRIMETHYL AND TRIPROPYL LEAD IN RATS GIVEN EITHER TRI- OR TETRA-ALKYL LEAD

Compound	Dose (mg./kg.)	Time Killed After Injection (hours)	Trialkyl Lead Compound Found (μg./g. or ml. wet wt.)			
			Blood	Liver	Kidney	Brain
Trimethyl lead chloride	15	4	160	40	23	2·25
	15	24	180	28·5	25·5	4·3
Tetramethyl lead	40	4	3·7	6·7	1·9	0
	40	24	22	10·2	1·9	0
Tripropyl lead chloride	10·5	2	0	10·7	3·3	—
	10·5	4	3·9	29·5	19·0	—
	10·5	24	1·1	20·0	19·0	—
Tetrapropyl lead	40	24	0	13·2	7·4	0·5

24 hours after intraperitoneal injection of the respective lead compounds is given in Table 3. The highest concentrations of trimethyl lead were found in the blood, but appreciable quantities were also present in liver and kidney. The amount in the brain was very low. In contrast to the trimethyl and triethyl lead (Cremer, 1959) only trace amounts of tripropyl lead were found in blood. The reason for this is not known, for, when trimethyl, ethyl, and propyl lead chlorides were added separately to whole rat blood *in vitro* over 90% of each compound was recovered from the red cells.

Conversion of Tetra to Trialkyl Leads in Rats

It was previously found that within four hours of injecting rats with tetraethyl lead large amounts of triethyl lead could be found in their tissues. *In vitro* studies showed that rat liver preparations were able to bring about a rapid conversion of tetra- to triethyl lead (Cremer, 1959).

When rats were killed four hours after an injection of tetramethyl lead and several tissues analysed for trimethyl lead only small amounts were found (Table 3). After 24 hours the amount of trimethyl lead in the blood and liver had increased but was still indicative of a fairly slow rate of conversion of tetramethyl lead compared with tetraethyl lead. Similarly, after giving rats 40 mg./kg. tetrapropyl lead the amount of tripropyl lead found in the tissues was less than that found after giving 10·5 mg./kg. of tripropyl lead chloride (Table 3).

Effect of Organo-lead Compounds on Slices of Rat-brain Cortex

Preparations of rat-brain cortex slices were incubated in Kreb's Ringer phosphate medium with glucose as substrate with and without added organo-lead compounds. Measurements were made of the oxygen consumption and the levels of lactate and pyruvate. The results are given in Table 4. Both trimethyl lead and tripropyl lead inhibited oxygen uptake, lowered the level of pyruvate and increased the level of lactate. This metabolic change indicated a block in the respiratory pathway whereby pyruvate formed from glucose is normally completely oxidized with the synthesis of high energy phosphate compounds. The glycolytic pathway of glucose catabolism was not inhibited so that lactic acid accumulated. The same type of inhibition of glucose metabolism had been found with triethyl lead. Neither tetramethyl nor tetrapropyl lead affected glucose metabolism by brain slices except when added in high concentrations (Table 4).

Activity of Brain Slices Prepared from Rats given Organo-lead Compounds.—It has already been shown that the glucose metabolism of brain slices given either tetra- or triethyl lead was inhibited in the same way as when triethyl lead was added *in vitro*. Brain slices prepared from rats given tripropyl lead showed a marked inhibition of glucose metabolism, but slices prepared from rats given tetra-

TABLE 4

EFFECT OF ALKYL LEAD COMPOUNDS ADDED *IN VITRO* ON THE METABOLISM OF SLICES OF RAT BRAIN CORTEX

Compound	Molar Concentration	% of Control		
		QO_2*	Lactic Acid	Pyruvic Acid
Trimethyl lead chloride	$8·8 \times 10^{-6}$	145	150	100
	$1·45 \times 10^{-5}$	73	350	57
	$5·8 \times 10^{-5}$	36	360	32
Tetramethyl lead	$1·5 \times 10^{-4}$	100	119	100
	$7·5 \times 10^{-4}$	100	139	100
Tripropyl lead chloride	$2·4 \times 10^{-7}$	116	170	100
	$4·8 \times 10^{-7}$	80	340	53
	$1·9 \times 10^{-6}$	41	240	32
Tetrapropyl lead	$7·0 \times 10^{-6}$	118	135	100

*Oxygen uptake.

Each flask contained 3·0 ml. of Kreb's Ringer phosphate solution with glucose 0·011 M, tissue slice of 50-60 mg. wet wt., and 0·2 ml. of 20% (w/v) KOH in the centre well for absorption of CO_2. The gas phase was 100% O_2. Incubation was at 37°C. for 75 minutes. QO_2 was calculated as μl. of O_2/mg. dry weight of tissue/hour. The control QO_2 value was 12·6. The control values for lactic acid and pyruvic acid were 179 μg. and 9·0 μg. respectively, estimated as the amount present in the medium after 75 minutes incubation.

TABLE 5

ACTIVITY OF BRAIN SLICES PREPARED FROM RATS GIVEN ALKYL LEAD COMPOUNDS

Compound	Dose (mg./kg.)	Time Killed After Injection (hours)	Brain Slices (% of Control)		
			QO_2	Lactic Acid	Pyruvic Acid
Trimethyl lead chloride	30	24	113	90	100
	30	48	100	110	120
Tripropyl lead chloride	10·5	2	98	96	115
	10·5	4	61	280	73
	10·5	19	49	314	51
Tetrapropyl lead	40·0	24	110	116	95

propyl lead varied only slightly from the controls (Table 5). No inhibition of glucose metabolism was found in slices prepared from rats given trimethyl lead even when the animals were showing definite signs of poisoning. This lack of effect might be largely accounted for by the high water solubility of trimethyl lead compared with triethyl or tripropyl lead so that on placing a brain slice in the incubation fluid the trimethyl lead content of the slice redistributes into the medium leaving an insufficient amount in the tissue to inhibit glucose oxidation. It is noticeable that the concentration of trimethyl lead required *in vitro* to inhibit brain slices is at least 30 times greater than that of tripropyl lead.

DISCUSSION

The signs of poisoning seen in rats after trimethyl lead chloride administration were very similar to those previously described for triethyl lead chloride poisoning (Cremer, 1959). The pattern of distribution of the two compounds in the tissues of the rats was also very similar. Tetramethyl lead was only toxic when given in relatively high doses; the signs of poisoning then resembled those seen in the rats given trimethyl or tetra or triethyl lead.

Since the hazard to man from these compounds is likely to occur in workers cleaning out petrol storage tanks or in manufacturing plants where the fumes and vapours might be inhaled the results of the inhalation studies on rats described in Table 2 are of particular interest. Tetramethyl lead given by this route had less than one-tenth of the toxicity of tetraethyl lead.

Tripropyl lead chloride provoked signs of poisoning differing in character from, and less well defined than, those seen in trimethyl or triethyl lead poisoned animals. The altered gait seen in many of these animals appeared to be a reversible phenomenon indicating a lack of permanent damage to the central nervous system which is consistent with there being no pathological changes seen on examination of the brains and spinal cords. As with the alkyl tin compounds (Barnes and Stoner, 1958) no permanent damage to cells of the central nervous system has been found in animals given any of the alkyl lead compounds studied in this laboratory.

Tetrapropyl lead, like tetramethyl lead was only toxic when given in relatively high doses.

The few experiments in which rabbits were used showed that they differed little from rats in their sensitivity towards the various alkyl lead compounds.

The most striking difference in the present results from those obtained using tetra and triethyl lead was that whereas tetraethyl lead was almost as toxic as triethyl lead (the LD_{50} values for rats were 15·4 and 11·2 mg./kg. respectively) both tetramethyl and tetrapropyl lead were considerably less toxic than the respective tri compounds.

Evidence was previously given (Cremer, 1959) in support of the hypothesis that tetraethyl lead itself is non-toxic but it is rapidly converted enzymically by the liver to triethyl lead which is toxic and to which metabolic processes of brain tissue are particularly sensitive. In the present work there is additional evidence in support of this conception which provides an explanation for the low toxicity of tetramethyl and tetrapropyl lead.

The results given in Table 4 show that both trimethyl and tripropyl lead, when added *in vitro*, inhibit the oxidation of glucose by slices of brain cortex. Neither tetramethyl nor tetrapropyl lead inhibited glucose oxidation at concentrations a hundred times greater than their trialkyl lead counterparts. Good agreement was previously found between the amount of triethyl lead found in the tissues of rats given just lethal doses of either tetra or triethyl lead which was indicative of a very rapid conversion of the tetra to the tri form. Results given in Table 3 show that the amount of trimethyl or tripropyl lead found in the tissues of rats given tetramethyl or tetrapropyl lead was very much less than that found after lethal doses of either tri compound. It thus seems highly probable that the low toxicity of tetramethyl and tetrapropyl lead is directly related to their slow rate of conversion. Pure samples of tetramethyl and tetrapropyl lead decompose at a slower rate than tetraethyl lead when stored. Tetramethyl lead is, however, highly explosive when ignited.

There have been several reports of human casualties suffering from tetraethyl lead poisoning (Cassells and Dodds, 1946; Boyd, Walker, and Henderson, 1957; Morrill, 1960). It is only recently that tetramethyl lead has been used as an additive to petrol and no reports of any human casualties from

contact with this compound have been found by the authors. From experimental work with laboratory animals it is suggested that the toxicity of the tetraalkyl lead compounds to a particular species is dependent upon the rate at which the species converts the tetra to the trialkyl lead form. Brodie (1956), in a review on the pathways of drug metabolism has pointed out that certain drugs are metabolized at completely different rates in animals compared with man. The metabolic pathway for a particular drug is, however, often the same for several different species. In the present work using rats and rabbits tetramethyl lead appears to be appreciably less toxic than tetraethyl lead but this may not be the case in man. The precautions taken in handling tetraethyl lead should be applied equally to tetramethyl lead.

The lower members of the trialkyl lead compounds are toxic to animals and their action appears to be predominantly on the central nervous system although in sub-lethal doses the changes brought about are not permanent.

We are grateful to Mr. D. Potter and Mr. Lynch for their skilled technical assistance, and to Mr. B. Purser and Mr. M. Wheeler who developed and carried out the analyses for the inhalation experiments.

References

Barnes, J. M., and Stoner, H. B. (1958). *Brit. J. industr. Med.*, **15**, 15.
Boyd, P. R., Walker, G., and Henderson, I. N. (1957). *Lancet*, **1**, 181.
Brodie, B. B. (1956). *J. Pharm. Pharmacol.*, **8**, 1.
Cassells, D. A. K., and Dodds, E. C. (1946). *Brit. med. J.*, **2**, 681.
Cremer, J. E. (1959). *Brit. J. industr. Med.*, **16**, 191.
Milner, G. W. C. (1957). *Principles and Applications of Polarography*. Longmans, Green, London.
Morrill, E. E. (1960). *Amer. industr. Hyg. Ass. J.*, **21**, 515.
Weil, C. S. (1952). *Biometrics*, **8**, 249.

Brit. J. industr. Med., 1961, **18**, 283.

A RAPID METHOD FOR THE MICRO-ESTIMATION OF LEAD IN URINE

BY

J. M. DICK, R. W. ELLIS, and J. STEEL

From the Nuffield Department of Industrial Health, King's College, Newcastle upon Tyne University of Durham

(RECEIVED FOR PUBLICATION JANUARY 2, 1961)

A method is described for the determination of microgram quantities of lead in small volumes of urine. Recoveries from urine samples of added lead show that the mean results of triplicate analyses will, 19 times out of 20, lie within $\pm 12{\cdot}5$ μg. per litre of the true concentration. The determination of lead in a single specimen of urine may be carried out in three hours.

In the literature on the estimation of urinary lead, it is usually recommended that not less than 100 ml. aliquots of the urine sample be taken for analysis. This has certain disadvantages, not the least of which is the time required to ash the organic content of the urine. In addition, spot samples of urine collected from groups of workmen are often less than the 200 ml. volume required for duplicate estimations. For these reasons, a rapid and accurate method was sought for the determination of total lead in small volumes of urine. The method finally devised is based on the dithizone mono-colour method described by Fischer and Leopoldi (1940).

The organic matter in a 20 ml. sample of urine is destroyed by ashing with nitric acid and the residue is dissolved in water. Bismuth is detected and removed by washing at *p*H 3·0-3·4 with a solution of dithizone in chloroform which is then discarded. Cyanide solution, buffered to *p*H 10, and an alkaline solution of dithizone are added and lead is extracted as the dithizonate into chloroform. Excess dithizone is washed out of the organic phase with potassium cyanide solution. The optical density of the washed chloroform extract is directly proportional to the quantity of lead present.

The advantages claimed for the method are as follows:

1. The small volume of urine needed for an accurate determination considerably reduces the time required to ash the sample and hence the time taken for the determination.

2. The ashing and extraction of lead with dithizone are carried out in the same vessel, thus reducing contamination from external sources.

Apparatus

The ashing and first extraction are carried out in a 50 ml. "Pyrex" glass conical flask fitted with a B19 socket. This flask is fitted with a B19 cone carrying a 2 mm. bore stopcock and 4 cm. stem (Fig. 1).

Excess dithizone is removed in a 150 ml. "Pyrex" separating funnel.

In order to render it lead-free, all glassware is treated with hot 50% nitric acid and rinsed with distilled water followed by lead-free water. The flasks should be kept covered at all times except during acid digestion, or when additions are made. Reagents are stored in "Polythene" wash bottles from which dropwise additions may conveniently be made. Where more accurate additions are required, the reagents are best stored in "Polythene" and borosilicate glass automatic dispensers. Spectrophotometric measurements are carried out on a "Unicam" SP600 spectrophotometer using 2 cm. glass cells.

Reagents

For details of methods for the preparation of lead-free reagents see the Report of the Analytical Methods Committee (1954).

Lead-free water.—Distilled water re-distilled in an all "Pyrex" glass apparatus.

Nitric Acid 0·5N.—Dilute 31 ml. concentrated nitric acid (B.D.H., low in lead) to 1 litre with lead-free water.

Hydrochloric Acid 1N.—Dilute 86 ml. concentrated hydrochloric acid (B.D.H., low in lead) to 1 litre with lead-free water.

Ammonium Hydroxide 5N.—Dilute 355 ml. concentrated 0·880 ammonium hydroxide (B.D.H., low in lead) to 1 litre with lead-free water.

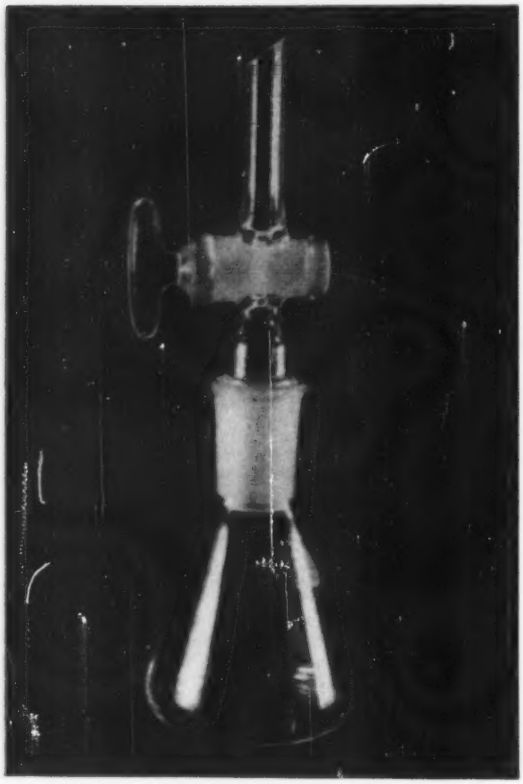


FIG. 1.—The ashing and extraction flask.

Chloroform.—Laboratory reagent chloroform stabilized by the addition of about 1% by weight of ethyl alcohol.

Dithizone Solution A.—Make up fresh each day. Dissolve 0·02 g. of pure diphenylthiocarbazone in 40 ml. of chloroform. To this solution add 4 ml. of 5N ammonium hydroxide, 36 ml. lead-free water, and shake for 30 seconds. Allow the phases to separate and reject the chloroform layer. The aqueous layer is dithizone solution A.

Dithizone Solution B.—Take 30 ml. of dithizone solution A and acidify with 1N hydrochloric acid until a permanent precipitate just forms. Add 20 ml. chloroform and shake for 30 seconds. Allow the phases to separate and reject the aqueous layer. The chloroform layer is dithizone solution B.

Ammonium Citrate Solution.—Dissolve 400 g. citric acid crystals in water and add sufficient reagent ammonia to make the solution alkaline to phenol red. Dilute the solution to 1 litre with water and make lead-free.

Potassium Cyanide 10% W/V Solution.—Dissolve 50 g. of potassium cyanide in sufficient water to make 100 ml., remove the lead content, and dilute the concentrated solution to 500 ml. with lead-free water.

Ammonium Citrate/Potassium Cyanide Solution.—Mix five parts of ammonium citrate solution with two parts of potassium cyanide solution. This solution is freshly prepared each day.

Methyl Orange Indicator Solution.—0·1% solution in lead-free water.

Phenolphthalein.—Dissolve 1 g. of phenolphthalein in 50 ml. of alcohol and add 50 ml. of lead-free water.

Antifoam.—Midland Silicone Antifoam "A"; 2% solution in carbon tetrachloride.

Standard Lead Solutions.—Dissolve 1·5984 g. of dried recrystallized lead nitrate in 1 litre of 1% nitric acid (10 ml. of nitric acid diluted to 1 litre with lead-free water).

One millilitre = 1·0 mg. Pb.

This solution keeps indefinitely in "Pyrex" glass or "Polythene" flasks.

Prepare a standard solution contining 1 μg. Pb per ml. by dilution of stock solution with 1% nitric acid. This dilute solution should be prepared as required.

Method

Acid Digestion.—Measure 20 ml. of urine into the 50 ml. flask, add two drops of Antifoam "A" and 1 ml. of concentrated nitric acid. If the urine collection contains a phosphate or carbonate deposit, add concentrated nitric acid (exactly 5% of the total urine volume), shake until the urine is clear, and withdraw 21 ml. aliquots for analysis. Insert the stopcock with the tap in the open position, and take the contents of the flask to dryness on the hot plate. Remove the flask from the hot plate in order to prevent ignition of the residue which will almost certainly produce a low result. Add 10 drops of nitric acid to the residue and again take to dryness. Repeat the operation once more so that a pure white residue is obtained. Dissolve the residue in 10 drops of acid and 10 ml. of lead-free water, bring the solution to the boil and allow to cool.

The solution should now be clear but occasionally it may be clouded due to the presence of silica which, however, does not interfere with the determination of lead.

Determination of Lead.—Add one drop of methyl orange indicator to the clear solution followed by sufficient 5N ammonia solution to produce a yellow colour. Adjust the *p*H of the solution to 3·0-3·4 by the addition of 0·5N nitric acid until a peach pink colour is produced. Add five drops of dithizone solution B and 5 ml. of chloroform. Shake the flask for 30 seconds, invert, and allow the contents to settle. The colour of the chloroform layer should be green showing excess dithizone, but if it is orange indicating the presence of bismuth, then add more dithizone solution B and repeat the process until the chloroform layer is coloured green. Discard the chloroform layer: this operation may be facilitated by warming the base of the flask with the hand.

Add three drops of phenolphthalein indicator to the solution followed by 1·4 ml. of ammonium citrate/potassium cyanide solution. Add 5N ammonium hydroxide until the *p*H of the solution reaches 10·0 as shown by the red colour of the indicator. Measure

carefully 10 ml. of chloroform into the flask followed by one drop of dithizone solution A. Shake the flask for 30 seconds and allow the layers to separate. If the chloroform layer is cherry red add another drop of dithizone solution A, shake the flask for 30 seconds, and again observe the colour of the chloroform layer. Repeat the addition of dithizone solution A until the colour of the chloroform layer is mauve, blue, or green showing the presence of excess dithizone.

Run the chloroform layer into a 150 ml. separating funnel containing 50 ml. of 0·5% potassium cyanide solution (2·5 ml. of 10% potassium cyanide solution made up to 50 ml. with lead-free water) and shake for 30 seconds. Dry the stem of the funnel with a rolled filter paper and plug lightly with cotton wool. Discard the first few drops of the chloroform layer and run the remainder of the layer into a 2 cm. spectrophotometer cell.

Measure the optical density of the chloroform solution at 520 mμ using chloroform as the reference solution. Carry out a blank determination with each series of tests.

Standard Curve.—Transfer aliquots of standard solution containing 0, 1, 2, 3, 4, and 5 micrograms of lead to 50 ml. conical flasks and make up the volumes to 20 ml. with lead-free water. Treat the solutions in exactly the same way as the urine samples for the development of colour and measure the optical density of the chloroform extracts at 520 mμ. Plot a graph of optical density against lead concentration.

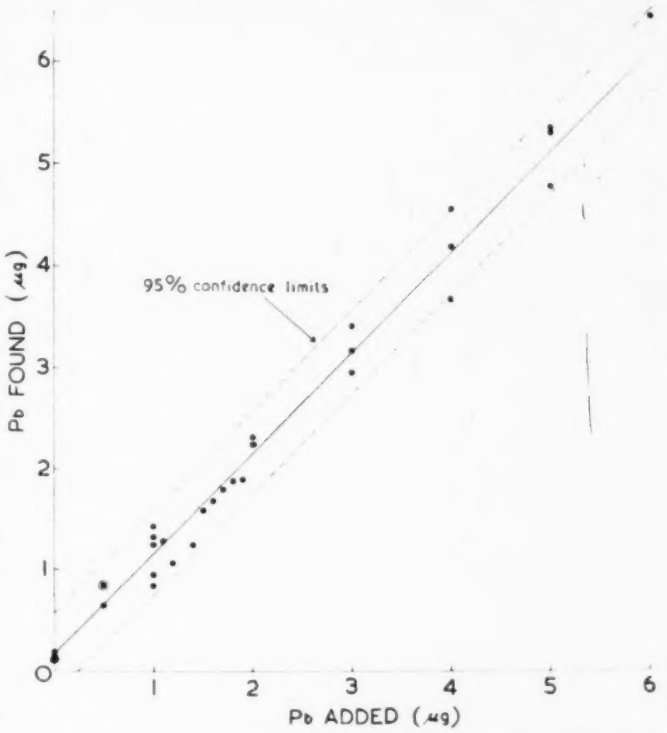


FIG. 2.—Recovery of lead added to urine samples.

Results

In order to check the method, recoveries of added lead were made from urine samples. To 33 aliquot portions of the same urine collection, amounts of standard solution containing from 0 to 6 μg. lead were added acurately from a micrometer syringe. Over that part of the range in which the upper limit for normal urine is found, namely 1 to 2 μg. Pb per 20 ml. or 50 to 100 μg. Pb per litre, standard solution was added to the urine aliquots in increments of 0·1 μg. lead. The lead contents of the urine samples were then determined by the method just described. The results are shown in the form of a graph of lead added plotted against lead found (Fig. 2).

The straight line of unit slope which is the best fit to the points on the graph was calculated and the 95% confidence limits drawn. It may be seen that 32 out of the 33 lead recoveries fall within these limits. The intercept on the *y* axis indicates that the concentration of lead originally present in the urine amounted to 0·15 μg. per 20 ml.

Confidence limits calculated for single, the mean of duplicate, and the mean of triplicate estimations show that 95% of results will fall within the range of ±21·7, ±15·3, and ±12·5 μg. per litre, respectively, of the true concentration of urinary lead. The mean value of triplicate analyses is thus considered to be sufficiently precise for the method to be used as an aid in the diagnosis of lead absorption or intoxication.

Discussion

Stannous tin, bismuth, and thallium, as well as lead, are extracted from alkaline cyanide solution by dithizone. Although these metals are infrequently present in urine, the following precautions were taken to prevent interference: (1) the nitric acid digestion converts any stannous tin to the stannic form which, in amounts usually present in urine, does not interfere; (2) bismuth is removed by dithizone extraction at *p*H 3·0-3·4; (3) the 0·5% potassium cyanide solution used to wash the chloroform extract removes any thallium, along with excess dithizone, from the chloroform layer.

In order to standardize the error due to the slight solubility of chloroform in potassium cyanide

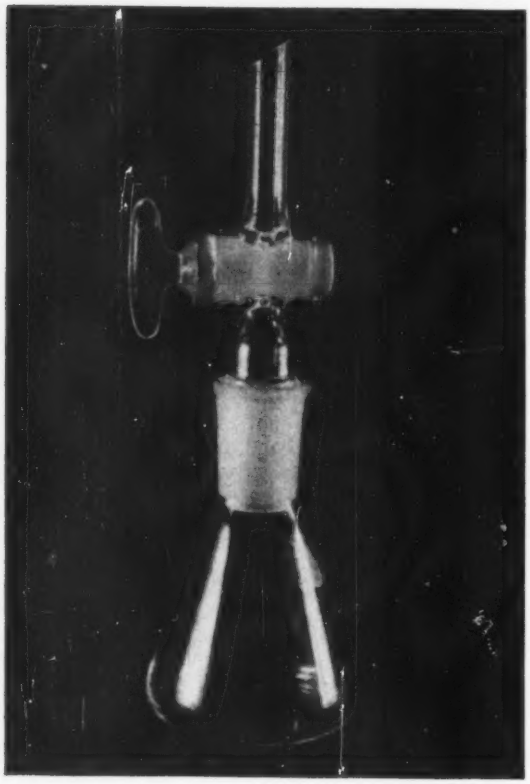


FIG. 1.—The ashing and extraction flask.

Chloroform.—Laboratory reagent chloroform stabilized by the addition of about 1% by weight of ethyl alcohol.

Dithizone Solution A.—Make up fresh each day. Dissolve 0·02 g. of pure diphenylthiocarbazone in 40 ml. of chloroform. To this solution add 4 ml. of 5N ammonium hydroxide, 36 ml. lead-free water, and shake for 30 seconds. Allow the phases to separate and reject the chloroform layer. The aqueous layer is dithizone solution A.

Dithizone Solution B.—Take 30 ml. of dithizone solution A and acidify with 1N hydrochloric acid until a permanent precipitate just forms. Add 20 ml. chloroform and shake for 30 seconds. Allow the phases to separate and reject the aqueous layer. The chloroform layer is dithizone solution B.

Ammonium Citrate Solution.—Dissolve 400 g. citric acid crystals in water and add sufficient reagent ammonia to make the solution alkaline to phenol red. Dilute the solution to 1 litre with water and make lead-free.

Potassium Cyanide 10% W/V Solution.—Dissolve 50 g. of potassium cyanide in sufficient water to make 100 ml., remove the lead content, and dilute the concentrated solution to 500 ml. with lead-free water.

Ammonium Citrate/Potassium Cyanide Solution.—Mix five parts of ammonium citrate solution with two parts of potassium cyanide solution. This solution is freshly prepared each day.

Methyl Orange Indicator Solution.—0·1% solution in lead-free water.

Phenolphthalein.—Dissolve 1 g. of phenolphthalein in 50 ml. of alcohol and add 50 ml. of lead-free water.

Antifoam.—Midland Silicone Antifoam "A"; 2% solution in carbon tetrachloride.

Standard Lead Solutions.—Dissolve 1·5984 g. of dried recrystallized lead nitrate in 1 litre of 1% nitric acid (10 ml. of nitric acid diluted to 1 litre with lead-free water).

One millilitre = 1·0 mg. Pb.

This solution keeps indefinitely in "Pyrex" glass or "Polythene" flasks.

Prepare a standard solution contining 1μg. Pb per ml. by dilution of stock solution with 1% nitric acid. This dilute solution should be prepared as required.

Method

Acid Digestion.—Measure 20 ml. of urine into the 50 ml. flask, add two drops of Antifoam "A" and 1 ml. of concentrated nitric acid. If the urine collection contains a phosphate or carbonate deposit, add concentrated nitric acid (exactly 5% of the total urine volume), shake until the urine is clear, and withdraw 21 ml. aliquots for analysis. Insert the stopcock with the tap in the open position, and take the contents of the flask to dryness on the hot plate. Remove the flask from the hot plate in order to prevent ignition of the residue which will almost certainly produce a low result. Add 10 drops of nitric acid to the residue and again take to dryness. Repeat the operation once more so that a pure white residue is obtained. Dissolve the residue in 10 drops of acid and 10 ml. of lead-free water, bring the solution to the boil and allow to cool.

The solution should now be clear but occasionally it may be clouded due to the presence of silica which, however, does not interfere with the determination of lead.

Determination of Lead.—Add one drop of methyl orange indicator to the clear solution followed by sufficient 5N ammonia solution to produce a yellow colour. Adjust the *p*H of the solution to 3·0-3·4 by the addition of 0·5N nitric acid until a peach pink colour is produced. Add five drops of dithizone solution B and 5 ml. of chloroform. Shake the flask for 30 seconds, invert, and allow the contents to settle. The colour of the chloroform layer should be green showing excess dithizone, but if it is orange indicating the presence of bismuth, then add more dithizone solution B and repeat the process until the chloroform layer is coloured green. Discard the chloroform layer: this operation may be facilitated by warming the base of the flask with the hand.

Add three drops of phenolphthalein indicator to the solution followed by 1·4 ml. of ammonium citrate/potassium cyanide solution. Add 5N ammonium hydroxide until the *p*H of the solution reaches 10·0 as shown by the red colour of the indicator. Measure

carefully 10 ml. of chloroform into the flask followed by one drop of dithizone solution A. Shake the flask for 30 seconds and allow the layers to separate. If the chloroform layer is cherry red add another drop of dithizone solution A, shake the flask for 30 seconds, and again observe the colour of the chloroform layer. Repeat the addition of dithizone solution A until the colour of the chloroform layer is mauve, blue, or green showing the presence of excess dithizone.

Run the chloroform layer into a 150 ml. separating funnel containing 50 ml. of 0·5% potassium cyanide solution (2·5 ml. of 10% potassium cyanide solution made up to 50 ml. with lead-free water) and shake for 30 seconds. Dry the stem of the funnel with a rolled filter paper and plug lightly with cotton wool. Discard the first few drops of the chloroform layer and run the remainder of the layer into a 2 cm. spectrophotometer cell.

Measure the optical density of the chloroform solution at 520 mμ using chloroform as the reference solution. Carry out a blank determination with each series of tests.

Standard Curve.—Transfer aliquots of standard solution containing 0, 1, 2, 3, 4, and 5 micrograms of lead to 50 ml. conical flasks and make up the volumes to 20 ml. with lead-free water. Treat the solutions in exactly the same way as the urine samples for the development of colour and measure the optical density of the chloroform extracts at 520 mμ. Plot a graph of optical density against lead concentration.

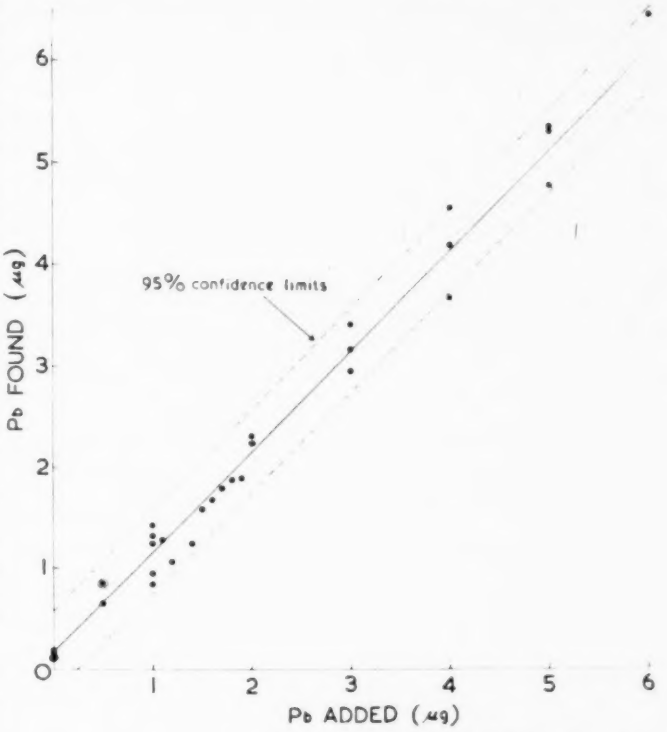


FIG. 2.—Recovery of lead added to urine samples.

Results

In order to check the method, recoveries of added lead were made from urine samples. To 33 aliquot portions of the same urine collection, amounts of standard solution containing from 0 to 6 μg. lead were added acurately from a micrometer syringe. Over that part of the range in which the upper limit for normal urine is found, namely 1 to 2 μg. Pb per 20 ml. or 50 to 100 μg. Pb per litre, standard solution was added to the urine aliquots in increments of 0·1 μg. lead. The lead contents of the urine samples were then determined by the method just described. The results are shown in the form of a graph of lead added plotted against lead found (Fig. 2).

The straight line of unit slope which is the best fit to the points on the graph was calculated and the 95% confidence limits drawn. It may be seen that 32 out of the 33 lead recoveries fall within these limits. The intercept on the *y* axis indicates that the concentration of lead originally present in the urine amounted to 0·15 μg. per 20 ml.

Confidence limits calculated for single, the mean of duplicate, and the mean of triplicate estimations show that 95% of results will fall within the range of ±21·7, ±15·3, and ±12·5 μg. per litre, respectively, of the true concentration of urinary lead. The mean value of triplicate analyses is thus considered to be sufficiently precise for the method to be used as an aid in the diagnosis of lead absorption or intoxication.

Discussion

Stannous tin, bismuth, and thallium, as well as lead, are extracted from alkaline cyanide solution by dithizone. Although these metals are infrequently present in urine, the following precautions were taken to prevent interference: (1) the nitric acid digestion converts any stannous tin to the stannic form which, in amounts usually present in urine, does not interfere; (2) bismuth is removed by dithizone extraction at *p*H 3·0-3·4; (3) the 0·5% potassium cyanide solution used to wash the chloroform extract removes any thallium, along with excess dithizone, from the chloroform layer.

In order to standardize the error due to the slight solubility of chloroform in potassium cyanide

solution, the volume of the cyanide wash was carefully controlled.

Because of the minute amounts of lead involved in the estimation, the elimination of sources of contamination is of the utmost importance. This is largely achieved by the use of plastic "squeeze" wash bottles as reagent dispensers. In this way contamination from pipettes, burette taps, and flask stoppers is avoided. Moreover, a considerable saving in time is effected by the use of dispensers rather than pipettes and this, taken together with the comparatively short time required to ash a 20 ml. sample, means that triplicate analyses of a single urine collection may be carried out in about three hours.

Parallel investigations of the mixed colour and reversion methods of lead estimation have shown that, under the conditions described above, the mono-colour method is to be preferred because of its higher sensitivity and lower variability.

The authors are grateful to Mr. D. J. Newell and Mrs. D. Weightman for statistical advice and calculations.

References

Fischer, H., and Leopoldi, G. (1940). *Z. anal. Chem.*, **119**, 161.
Report of the Analytical Methods Committee (1954). *Analyst*, **79**, 397.

Brit. J. industr. Med., 1961, **18**, 287.

THE DISTRIBUTION AND EXCRETION OF INHALED MERCURY VAPOUR

BY

J. C. GAGE

From Imperial Chemical Industries Limited, Industrial Hygiene Research Laboratories, The Frythe, Welwyn, Herts

(RECEIVED FOR PUBLICATION FEBRUARY 22, 1961)

Rats have been exposed for varying periods to an atmosphere containing 1 mg./cu.m. mercury vapour. The toxic effects produced showed resemblances to signs of mercurialism in man. An attempt has been made to study the kinetics of absorption and excretion of mercury from measurements of the amounts excreted and stored in the tissues.

The efficiency of absorption of mercury by the rat lung is about 50%. A small proportion is excreted into the gut. After about 10 days of continuous exposure a steady state is reached in which excretion balances absorption. During short exposures the turnover of mercury in all tissues except brain is fairly rapid and most of the mercury is cleared from the body within a week after exposure. The urinary excretion of mercury, during the initial stage of storage in the tissues and the final stage of clearance, shows divergencies from the simple exponential pattern; there appears to be a delay mechanism in the kidney which, in intermittent exposures, may result in the occurrence of peak excretion during periods of non-exposure. After more prolonged exposures the mercury in the kidney appears to be converted to a form which is only very slowly excreted.

The significance of the urinary excretion of mercury by man after industrial exposure to mercury vapour is discussed. The rat experiments suggest that single measurements will give only limited information concerning industrial conditions, but that an approximate assessment of the total absorbed during a working week would be obtained if it were possible to make a seven-day collection of urine. Repeated measurements after exposure would yield information on the duration of exposure and would have some diagnostic value.

The quantitative determination of mercury in urine admits considerable sensitivity and specificity and numerous attempts have been made to utilize such measurements in the control of human industrial exposure to mercury vapour. A review on this subject by Heyroth (1949) is not, however, very encouraging and the opinion of most investigators appears to be well summarized by his comment that the elimination of mercury in the urine is too irregular to be of value in determining the severity of occupational exposure, and its absence from urine in occasional typical cases of mercury poisoning makes a search for it of uncertain value as a means of determining that exposure has occurred previously. More recent investigations have indicated some measure of correlation between urinary excretion of mercury and signs of mercury poisoning, but very wide individual variations in excretion obscure the interpretation of urine analysis (Turrian, Grandjean, and Turrian, 1956; Totušek and Waelschová, 1954).

A study of the earlier literature tends to confirm Heyroth's opinion that uncertainty regarding the validity of the analytical methods renders much of the work of doubtful significance. Modern methods are more reliable and although it is still possible for errors to be introduced, it seems unlikely that the present uncertainty concerning the interpretation of urinary mercury concentrations can be attributed to analytical errors. It seems more probable that the excretion of mercury follows a complex pattern which has not yet been revealed by experimental studies on man or animals, and a simple interpretation cannot be based on the analytical results.

The first step in elucidating the excretion of mercury in man would be a detailed knowledge of the fate of inhaled mercury in an experimental

animal. Extensive investigations have been undertaken by Ashe, Largent, Dutra, Hubbard, and Blackstone (1953), in which a variety of species was exposed to known mercury concentrations in air, but their experiments were directed primarily to the establishment of a threshold concentration for the appearance of the toxic effects. The protocols of these experiments yield only limited information concerning the balance between mercury absorbed and mercury eliminated and the rates of accumulation and excretion. A study of the kinetic aspects of the fate of inhaled mercury has, therefore, been undertaken, using the rat as the experimental animal; observations have also been made on the progress of signs of mercury poisoning under controlled conditions.

Preparation of Mercury Vapour Atmospheres

A current of nitrogen (0·3 l./min.) was passed over the surface of 100 ml. mercury contained in a 500 ml. round-bottom flask, heated by means of an electric mantle to 180°C. The nitrogen was then passed through a filter thimble to remove any condensed mercury droplets and diluted 15 to 20-fold with clean air. This dilution was passed through a vessel in which it was mixed by turbulence; it was then daily analysed by the method described below and the air-flow adjusted if necessary to give a concentration of 1·0 ± 0·1 mg./cu.m. mercury.

The mercury concentration in the atmosphere was determined by absorption in acid-permanganate followed by a modification of the dithizone titration method of Fabre, Truhaut, and Boudène (1958). A 2-litre sample of the atmosphere was aspirated at a constant speed of 1 l./min. through a bubbler containing a mixture of 8 ml. 6% w/v aqueous potassium permanganate and 2 ml. 50% v/v aqueous sulphuric acid. The solution so obtained was decolorized with 2 ml. 20% aqueous hydroxylamine hydrochloride solution, and 2 ml. 10% w/v aqueous urea solution and 1 ml. 2·5% w/v ethylenediamine tetra-acetic acid disodium salt solution were added. A control solution was prepared by mixing the same amounts of all of the above reagents in a tube similar in shape to that used in the bubbler. Both solutions were adjusted to *p*H 1·5, using external indicator paper and a solution of sodium acetate prepared by mixing the crystalline salt with an equal weight of water. A dilute dithizone solution was prepared by diluting a stock 20 mg.% solution in carbon tetrachloride 1 to 20 with chloroform; the test solution was titrated with this dithizone solution, added in small portions from a burette followed by vigorous shaking of the stoppered tube, until the separated lower organic layer developed a colour intermediate between the green of dithizone and the orange of the mercury-dithizone complex. The volume of dithizone solution required for the test solution was noted and the same volume added to the control tube. The latter was then titrated with a standard aqueous solution containing 1 μg./ml. mercury, added in small portions followed by vigorous shaking, until by visual comparison the colours of the separated organic layers in the two tubes were identical. The titration figure gave the mercury content of the air sample expressed in μg. Analytical quality reagents were used in this determination. The efficiency of this method of analysis was demonstrated by making an estimation with a second bubbler containing acid permanganate in series with the first; no mercury could be detected in the second bubbler.

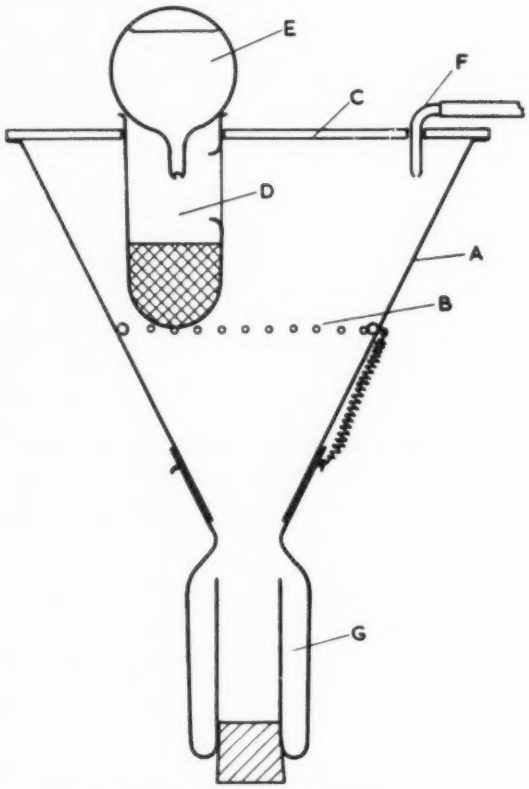


FIG. 1.—Diagram of cage for exposing individual rats to mercury vapour. A, polyethylene funnel; B, stainless steel wire grid; C, "Perspex" lid; D, feeding tube; E, water bottle; F, inlet for mercury atmosphere; G, separator for collecting urine and faeces.

Exposure of Experimental Animals

Albino Wistar strain female rats, average initial weight 160 g., were housed in the individual metabolism cages shown in Fig. 1. They were supported on a stainless steel wire grid inserted in a 20 cm. diameter polyethylene funnel covered with a "Perspex" lid through which passed the glass feeding tube and a water bottle. The feeding tube was 4 cm. diameter and 8 cm. long and contained a one-day supply of ground rat cube moistened with water to a paste, and it was pierced with a 3 cm. diameter hole bearing an internal flange which was sufficiently large to permit the entry of a rat's head

TABLE 1

RECOVERY OF MERCURY ADDED TO NORMAL RAT TISSUES AND EXCRETA; ALL SAMPLES CONTAINED INITIALLY NEGLIGIBLE AMOUNTS OF MERCURY

Material	Amount Taken for Analysis (g.)	μg. Mercury		% Recovery
		Added	Found	
Kidney	2	4	3·7, 3·8	94
Liver	2	4	3·9, 3·9	97
Spleen	2	4	3·9, 3·9	97
Brain	2	4	3·8, 3·9	96
Lung	2	4	3·6, 3·7	94
Gut	2	4	3·8, 3·8	95
Blood	2	4	3·7, 3·8	94
Urine	10 ml.	4	3·9, 3·9	97
Faeces	2	4	3·0, 3·4	80

TABLE 2

MERCURY CONTENT OF TISSUES, EXPRESSED IN μg. PER RAT, AFTER 28 DAYS CONTINUOUS EXPOSURE TO 1 mg./cu.m. MERCURY (AVERAGE OF FOUR RATS)

Tissue	Period After Exposure (days)		
	0	2	4
Lungs	17·5	8·2	6·7
Liver	25	5·1	2·3
Kidneys	222	155	137
Spleen	0·8	0·6	0·2
Brain	9·8	13·8	13·3
Gastro-intestinal tract	12·8	2·7	3·7
Blood (estimated)*	4·4	3·0	1·4
Muscle (estimated)†	11·4	11·3	0
Total	304	200	165

*Calculated assuming 10 ml. blood per rat.
†Calculated assuming 43% body weight.

without allowing it to withdraw any of the diet. Rats exposed in these cages to a mercury-free atmosphere for five days a week showed a weight increase over a period of seven weeks similar to that of rats kept under normal conditions, though during the first few weeks the weight gain while in the metabolism cage was less than normal, with recovery over the week-ends. This could be correlated with an initial reduced food intake in the metabolism cage.

The test mercury atmosphere passed through a manifold to each of six metabolism cages, entering through a hole in the lid. The excreta were collected in a glass separator shown in Fig. 1, constructed from two concentric tubes, the faeces falling in the inner tube and the urine trickling down the funnel and collecting in the annular outer chamber. This type of separator has been in use for many years in this laboratory and has the advantages of robustness and simplicity of construction; if required it may be placed in a vacuum flask containing solid carbon dioxide which freezes the urine as soon as it enters the separator.

Analysis of Excreta and Tissues

Oxidation with Acid Permanganate.—The urine was well mixed, diluted if necessary, and 10 ml. were measured into a glass-stoppered tube. To this were added 2 ml. 50% v/v sulphuric acid, 0·5 g. potassium permanganate and a few drops of octanol. The tube was heated in a boiling water bath, with the stopper loosely inserted, for 30 minutes. A 2% homogenate of the total daily faeces was prepared in water by means of a macerator and a 100 ml. portion was measured into a 250 ml. round-bottomed flask and 10 ml. sulphuric acid, 5 g. potassium permanganate, and a few drops of octanol were added. A water-cooled condenser was attached by means of a standard-taper joint and the contents of the flask were boiled on an electric mantle for three hours. The procedure used for tissues was similar to that for faeces, except that a 5% homogenate was prepared. The control solution was prepared by heating to the boiling point a volume of acid-permanganate equal to the volume of homogenate used, decolorizing with hydroxylamine hydrochloride, and processing as above.

Determination of Mercury Content.—A slight excess of solid hydroxylamine hydrochloride was added to the hot solution after permanganate oxidation which was then filtered if necessary. After cooling, all or part of the solution was taken and the analysis completed as described above for the determination of mercury in the test atmosphere. Recoveries of known amounts of mercury added to normal rat tissue and excreta are shown in Table 1.

Experimental Results on Rats

Continuous Exposure.—A group of 12 rats was exposed 24 hours a day, for a period of 28 days to the 1 mg./cu.m. concentration of mercury vapour. All the rats survived this exposure but showed a loss in weight over the period. The rats became lethargic by the seventh day and at the termination of the exposure they were showing slight tremors when lifted by their tails. Pooled urine and faeces were collected from the rats each day; at the end of the exposure period four of the rats were killed, and at intervals of two days the remaining rats were killed in groups of four. Tissues were removed from these rats for mercury analysis. The results obtained are shown in Table 2; the figures for blood and muscle are based on the assumptions that each rat contained 10 ml. blood and that the skeletal muscle constituted 43% of the body weight (Donaldson, 1924). The whole gastro-intestinal tract was removed and washed through with water before analysis. The daily excretion of mercury in urine and faeces by these rats is shown in Fig. 2.

In order to study the retention and excretion of mercury in the initial stages of continuous exposure, the experiment was repeated with a group of 12 rats. Urine was collected each day and at two-day intervals four of the rats were killed and the kidneys removed for mercury determinations. The results of this experiment are shown in Table 3.

Repeated 100-hour Exposures.—A group of six rats was exposed to the 1 mg./cu.m. mercury vapour atmosphere continuously for 100 hours from Monday to Friday in each week, for six successive

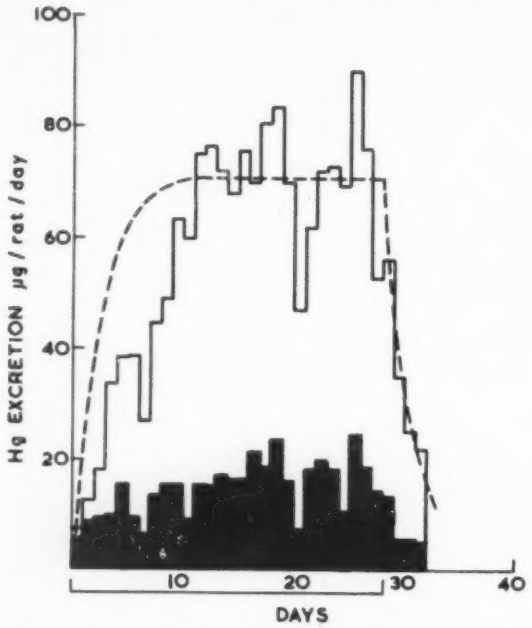


FIG. 2.—Total excretion of mercury after 28 days of continuous exposure to 1 mg./cu.m. mercury. Square bracket indicates period of exposure. Faecal excretion indicated by black area. The broken line is the curve calculated according to equations (3) and (5).

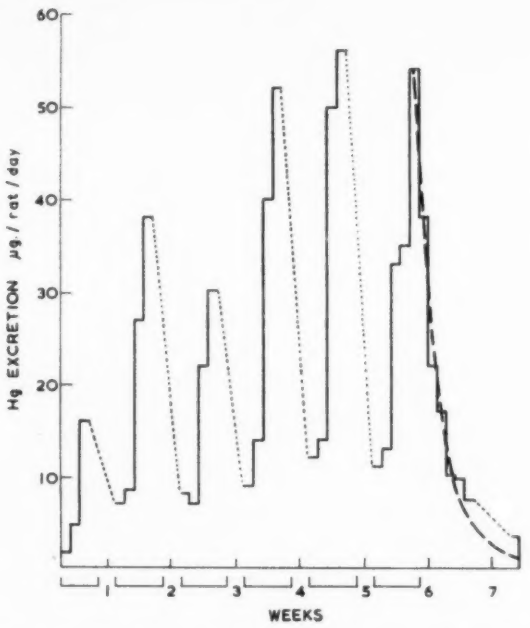


FIG. 3.—Urinary excretion of mercury after repeated 100-hour exposures to 1 mg./cu.m. mercury in successive weeks. Square brackets indicate periods of exposure. Dotted lines are periods when excretion was not measured. Broken line is the curve calculated according to equation (3).

weeks. Growth during this period was retarded, the average increase per rat being about 10 grammes. By the third week of exposure the rats were in poor condition; they appeared lethargic and they squeaked when touched. After five weeks all the rats were showing fine tremors which were more noticeable when the animals were lifted by their tails. The tails themselves had an unusual rigidity.

The pooled urine was analysed for mercury four times a week, and occasional daily analyses were made during the week-ends when the rats were not exposed to the mercury atmosphere. The results are shown in Fig. 3. Two of the rats were killed at the end of the exposure, two more three months later, and the two survivors were killed after a further three months. The mercury contents of the organs are shown in Table 4. Urine was collected from the survivors for a time after the exposure and the mercury contents are included in Fig. 3. The tremors which were observed during the exposure periods soon disappeared from the survivors but they continued to squeak when touched for several weeks. During the first weeks of the exposure the urine showed a fluctuating protein content, reaching a maximum of 0·9 mg./ml. Later during the experiment the protein excretion became very small.

TABLE 3

URINARY EXCRETION AND KIDNEY STORAGE OF MERCURY DURING INITIAL STAGE OF CONTINUOUS EXPOSURE TO 1 mg./cu.m. MERCURY (AVERAGE OF FOUR RATS)

Duration of Exposure (days)	Mercury Content (µg.)		a
	Urine	Kidney	
1	1·8		
2	6·5	72·5	0·09
3	19		
4	50	115	0·43
5	52·5		
6	53·5	142	0·38

TABLE 4

MERCURY CONTENT OF ORGANS FROM RATS EXPOSED TO 1 mg./cu.m. MERCURY FOR SIX WEEKLY 100-HOUR PERIODS; AVERAGE OF TWO RATS, EXPRESSED AS µg./RAT

Period after Exposure (months)	Organs				
	Lungs	Liver	Kidney	Spleen	Brain
0	10	4·6	137	0	8
3	0	0	15	0	1·6
6	0	0	1·9	0	1·4

TABLE 5

INDIVIDUAL WEEKLY MERCURY EXCRETION IN URINE OF SIX RATS EXPOSED TO 1 mg./cu.m. MERCURY FOR 100 HOURS/WEEK, FOR SIX WEEKS

Week	Mercury Excretion (μg./Rat)						Mean	Coefficient of Variation (%)
1	27·3	29·9	29·9	26·9	47·2	13·6	29·1	36·8
2	86·0	92·0	76·8	90·5	103	53·1	83·6	20·5
3	105·6	111·8	81·9	64·2	81·2	69·9	85·8	15·5
4	140·1	190	128·9	140·5	233·7	167·3	166·6	23·8
5	221·5	246·3	220·1	203·8	218·7	222·5	222·1	6·2
6	166·5	227·7	175·6	140·6	153·8	178·9	173·8	17·2

The experiment was repeated with six rats maintained in the same mercury atmosphere for 100 hours a week for seven weeks. The rats showed toxic signs similar to those described in the previous experiment and two died during the seventh week. Death was preceded by loss of weight, fall of body temperature, and coma. Necropsy and histopathological examination showed congestion of the lungs and livers, and degeneration of the cortical tubules of one rat. Individual urine collections were taken from these rats during the exposure period, and the weekly totals of each rat are shown in Table 5, together with the coefficients of variation of these totals round the weekly means.

Repeated Seven-hour Exposures.—Six rats were exposed to 1 mg./cu.m. mercury for seven hours a day, five days a week, for 18 successive weeks. The rats showed a slight increase in weight during this period but their rate of growth was much less than that of rats kept under similar conditions but in the absence of mercury vapour. After the seventh week the rats appeared lethargic and two squeaked when touched; by the fifteenth week these two rats showed fine tremors when lifted by their tails. At the end of the exposure period two rats were killed for organ analysis and the survivors soon returned to good condition and started to increase in weight more rapidly. Pooled urine was collected during and after exposure as described above, and analytical figures for urinary mercury are shown in Fig. 4. There was a slight fluctuating excretion of protein in urine with a maximum of 0·2 mg./ml. The surviving rats were killed at two and four months after exposure and the organ analyses are shown in Table 6.

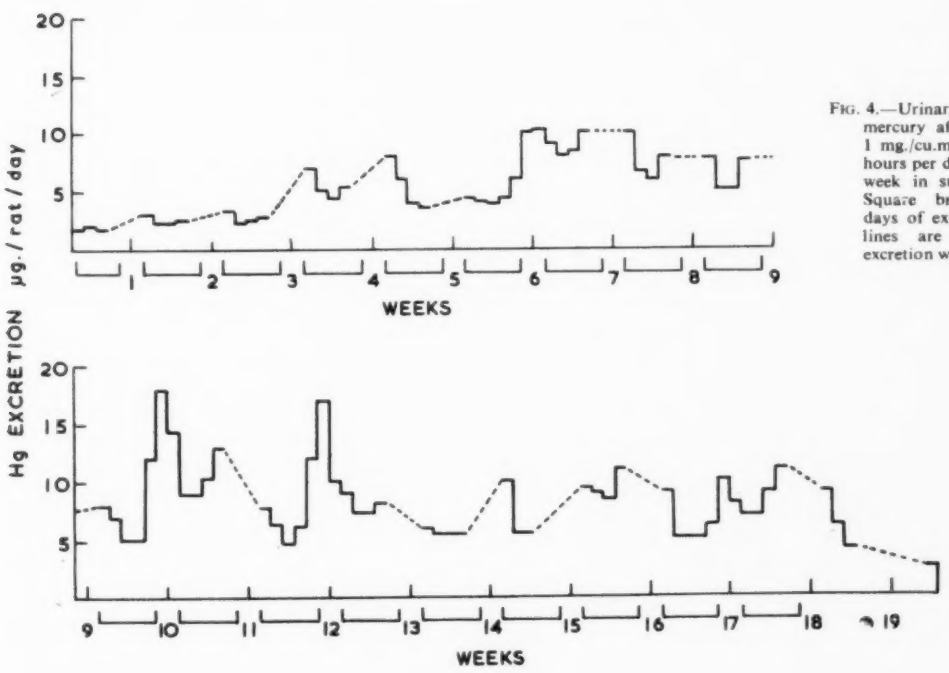


FIG. 4.—Urinary excretion of mercury after exposure to 1 mg./cu.m. mercury seven hours per day, five days per week in successive weeks. Square brackets indicate days of exposure. Dotted lines are periods when excretion was not measured.

TABLE 6

MERCURY CONTENT OF ORGANS FROM RATS EXPOSED TO 1 mg./cu.m. MERCURY SEVEN HOURS A DAY, FIVE DAYS A WEEK, FOR 18 WEEKS; AVERAGE OF TWO RATS EXPRESSED AS μg./RAT

Period after Exposure (months)	Organs				
	Lungs	Liver	Kidney	Spleen	Brain
0	1·0	0·7	140	0·1	5·3
2	0	0	14·2	0	1·9
4	0	0	0	0	1·3

Discussion

The excretion of mercury by rats exposed to an atmosphere containing 1 mg./cu.m. mercury for four weeks, is shown in Fig. 2 and can be considered in three stages. During the first 10 days there is a progressive increase in the mercury excreted; this is followed during the remaining 18 days of exposure by a daily excretion fluctuating around a mean value, and after the termination of the exposure the excretion rapidly falls. The major contribution to this pattern of excretion is made by the urine and the faecal excretion shows less variation. It seems unlikely that the average of 15 μg. mercury per day in the faeces derives from ingestion and it may be attributed to an excretion into the gut.

The first phase of the excretion shown in Fig. 2 indicates a progressive accumulation of mercury in the body, while the central plateau corresponds to a "steady state" in which the body is saturated and absorption is equal to excretion. If the respiratory minute volume of the rat is taken to be 100 ml. (Gaddum, 1959), the total mercury content of the air inspired in 24 hours is in the region of 140 μg.; as the average daily excretion during the saturation stage is about 71 μg., the efficiency of retention of mercury by the lungs is approximately 50%. Published figures for the retention of mercury in the lungs by man and the dog range between 15 and nearly 100% (Storlazzi and Elkins, 1941); in this laboratory experiments with isolated cat lungs artificially perfused with whole blood and ventilated with air containing 1 mg./cu.m. mercury, have shown that between 39 and 66% was retained by the lungs (Swan, 1960). It seems unlikely that the considerable fluctuation round the average value during the saturation stage can be attributed to a periodic variation in the efficiency of retention of mercury by the lungs, and it may derive from changes in the mercury retention by the body, or more probably in the magnitude of the respiratory minute volume. Adamo (1939) has shown that the increased respiratory volume attendant on an increased carbon dioxide content or humidity of the inspired air resulted in a greater absorption of mercury. This factor may also be a major contribution to the variation in the mercury excretion of individual rats exposed to 1 mg./cu.m. mercury for repeated 100-hour periods, which is shown in Table 5.

The total accumulation of mercury in the tissues during the first stage of the continuous exposure experiment shown in Fig. 2 can be estimated from the difference between the total excreted during this period and the total absorbed. If the daily absorption is assumed to be 71 μg. the figure so obtained is 370 μg. A direct determination of the amounts stored can also be obtained from the tissue analysis in Table 2; the first entry in the Table gives a total of 304 μg. which is in sufficiently good agreement with the figure quoted above, bearing in mind the errors involved, to make it likely that no significant reservoir of mercury has been missed.

At the termination of the continuous exposure the excretion of mercury does not immediately cease, due to a release of mercury stored in the tissues. During the first two-day period after exposure the body loses 104 μg. and during the second, 35 μg.; these figures agree well with the total excretion of 90·5 and 47·6 μg. during the same two periods (Fig. 2). As the bulk of the stored mercury is found in the kidneys, and most of the excreted mercury in the urine, the kinetics of excretion may be based on the assumptions that the daily loss from the body appears in the urine, and that this loss is proportional to the amount of mercury in the kidneys. The following equations may be written where v is the amount in the kidneys, u is the quantity of mercury excreted in urine in one day, u_o is the initial value, and α is a constant.

$$-dv/dt = u = \alpha v \quad (1)$$

$$u = u_o e^{-\alpha t} \quad (2)$$

$$\log_{10} u = \log_{10} u_o - \alpha t/2{\cdot}3 \quad (3)$$

The nature of the post-exposure excretion is seen more clearly in Fig. 3; it approximates to the exponential pattern expected from equation (2) and the broken line in this Figure has been calculated from equation (3) with the constant α given the empirical value 0·4/day.

During the initial period when mercury is accumulating in the tissues, the daily excretion of mercury in the urine is given by equations (4) and (5), where α has the same value 0·4 and u_o is the average daily excretion when saturation of the tissues is achieved.

$$u = u_o (1 - e^{-\alpha t}) \quad (4)$$

$$\log_{10}(u_o - u) = \log_{10} u_o - \alpha t/2{\cdot}3 \quad (5)$$

The broken line in Fig. 2 is calculated on the assumption that the total excretion can be represented by equations (3) and (5), and $u_o = 71$ μg./day. The experimental post-exposure values agree well with this curve, but the values at the commencement

of the exposure do not show the expected initial rapid increase; this deviation is more marked if the urinary excretion alone is compared with the theoretical curve with $u_o = 56$. The urinary excretion in Fig. 2 shows a slow increase for the first few days followed by a more rapid rise; this tendency is also seen in the urinary excretion at the commencement of each weekly exposure period shown in Fig. 3, and it is still more marked in Fig. 4. This initial delay in excretion is not due to a delay in the accumulation of mercury in the kidney, for the analytical figures in Table 3 indicate a rapid increase in the amount of mercury in the kidney during the first few days of exposure. This Table also includes the values of the constant α from equation (1) during this period, calculated from the ratio between the amount in the kidney and the corresponding daily output in the urine. The value of α on the second day of exposure is much less than 0·4 and only reaches this value on the fourth day; this indicates that initially the mercury in the kidney is excreted more slowly and that the simple assumptions in equation (1) are inadequate completely to define the kinetics of mercury excretion.

It may be seen from Fig. 3 that when rats are exposed to 1 mg./cu.m. mercury for successive 100-hour periods, the peak urinary mercury excretion occurs at the end of the week and rises progressively until after four weeks it has reached an approximately constant value which is of the same order as the "steady state" urinary excretion in Fig. 2. This suggests that the tissues are then saturated and the mercury lost during the week-end when the rats are not exposed to mercury vapour is regained during the subsequent exposure. However, the results obtained from rats exposed for five daily seven-hour exposures each week (Fig. 4), show that the excretion of mercury in urine is more complex than this. In these experiments the values were found to decrease from Monday onwards and start again to show an increase only on the following Friday. When excretion measurements were made over the week-ends the peak excretion was found to occur during this period of non-exposure, this peak approaching the value of approximately 20 μg. which would be expected if the tissues were saturated. This observation suggests that the excretion of mercury does not immediately start to decrease when exposure ceases and that the unexpected excretory pattern is due to the combination of an initial delay in the rise of excretion and a terminal delay in the fall-off.

Tables 4 and 6 show that mercury can still be found in the kidneys up to six months after exposure. This is not to be expected from equation (3) which suggests that 90% of the mercury should be cleared within six days. This suggests that another mechanism involving a slower clearance from the kidney is also involved, and there is some indication of this in the terminal excretion of mercury in Fig. 2 where the lower values begin to diverge from the expected exponential curve. From Tables 4 and 6 the average loss from the kidney during the period three to six months after exposure is 1 to 2% per day, an amount too small to be measured with certainty in urine. The clearance of mercury from brain is even slower than from kidney and there can be no doubt that the tremors and hypersensitivity which were exhibited by the rats in these experiments were due to the affinity of the central nervous system for mercury. There are interesting analogies between these effects on rats and the reported signs of mercurialism in man.

It is evident from the investigation that the fate of inhaled mercury vapour is complex. The lungs seem to be a fairly efficient means of extracting the vapour from the air; it seems probable that a chemical reaction at the surface of the lungs is involved in the retention of the fraction absorbed as there is no evidence that the solubility of metallic mercury in the body fluids is high. The lung analyses shown in Table 2 indicate that the mercury is fairly rapidly cleared from the lung, and this is confirmed from other sources (Wirth, 1936). A small proportion of the mercury is excreted from the circulating blood through the gut but the bulk is taken out by the kidney, from which most is excreted within a week. Much less mercury is found in other tissues and its rate of clearance from all is rapid with the exception of brain where the excretion is very slow.

The slower clearance of mercury from the kidney at the commencement of exposure suggests that mercury arriving at the kidney is not immediately excreted but must first undergo a change of state or location. The peak urinary excretion which may occur after interruption of exposure suggests that this transformation or translation of mercury within the kidney is independent of the contemporaneous supply of mercury to the kidney, and that the excretory pattern refers to the absorption a few days previously. A different mechanism must be postulated for the slow excretion which still occurs after exposure; this may be due to an irreversible incorporation of mercury into the cell proteins and the rate of excretion may be connected with the metabolic turnover of protein. The establishment of these hypotheses must wait upon a detailed chemical and cytological study of the distribution of mercury in the kidney and its state of chemical combination in the kidney and in urine.

On the basis of these observations it is possible to

speculate on the significance of urinary mercury measurements in the control of human exposure to mercury vapour. It has been suggested that after short-term exposure the mercury appearing in urine is related to the amount in the kidney which has been inhaled a few days previously. A single measurement of mercury in urine is not, therefore, related to the immediate atmospheric concentration, and in order to obtain an estimate of the total amount absorbed during a working week it is necessary to collect a seven-day sample including the week-end. If the results obtained with rats at 1 mg./cu.m. can be applied to man breathing 10 cu.m. a day for five days of an atmosphere containing the maximal allowable concentration of 0·1 mg./cu.m., then the daily absorption might be expected to be in the region of 500 μg., with a daily excretion during the working week well below this figure but with a total excretion over seven days of approximately 2·5 mg. If the total exposure had been of short duration the bulk of the mercury would be eliminated in a few days after exposure. The rat experiments have also indicated that if exposure to mercury vapour is maintained for several weeks there is a progressive conversion of the mercury in the kidney to a slowly diffusible form and it is conceivable that prolonged human exposure will result in the accumulation in the kidney of mercury in this type of combination, resulting in an excretion which is maintained for a considerable period after exposure and which may amount to several hundred μg. per day. It does not seem necessary to postulate wide individual differences in the ability to absorb and retain mercury in order to account for the reported differences in the urinary excretion of men exposed to similar industrial conditions for it seems that much of this can be attributed to the complex nature of mercury excretion. The lack of a simple correlation between mercury excreted in urine and the mercury concentration in the atmosphere inhaled suggests that a threshold concentration for mercury in urine cannot be derived from an accepted maximal allowable concentration for mercury in air.

The application of urinary mercury determinations to assess the amount of mercury in the body, or to confirm a diagnosis of mercury poisoning, is also likely to be difficult. Studies of human excretion of mercury have led to the conclusion that a high value is of diagnostic significance only when symptoms and signs of poisoning can be demonstrated (Bidstrup, Bonnell, Harvey, and Locket, 1951). The mercury appearing in urine is related to the mercury content of the kidney and there is no reason to suppose that mercury located there can harm any organ other than the kidney itself. More information would be provided by a knowledge of the state of combination of mercury in the kidney, as this might enable an assessment of the duration of exposure to be made which would indicate whether there had been an opportunity for mercury to enter the central nervous system. At present the only way of investigating this appears to be by repeated measurements of the excretion in urine after exposure has ceased, but the interpretation of the results so obtained requires more knowledge of the kinetics of human excretion. Another approach would be through a qualitative study of urine, if it could be shown that the transient and slow excretions were reflected in different states of chemical combination of mercury in urine.

I am indebted to Dr. J. G. S. Crabbe for a pathological report on the tissue sections, and to Mr. Z. S. Berczy for skilled technical assistance.

References

Adamo, M. (1939). *Rass. Med. industr.*, **10**, 684.
Ashe, W. F., Largent, E. J., Dutra, F. R., Hubbard, D. M., and Blackstone, M. (1953). *Arch. industr. Hyg.*, **7**, 19.
Bidstrup, P. L., Bonnell, J. A., Harvey, D. G., and Locket, S. (1951). *Lancet*, **2**, 856.
Donaldson, H. H. (1924). "The Rat", 2nd ed. *Mem. Wistar Inst. Anat.*, No. 6. Philadelphia.
Fabre, R., Truhaut, R., and Boudène, C. (1958). *Ann. Biol. clin.*, **16**, 286.
Gaddum, J. H. (1959). *Pharmacology*, 5th ed. Oxford University Press, London.
Heyroth, F. F. (1949). *Industrial Hygiene and Toxicology*, vol. II, ed. F. A. Patty. Interscience Publ., New York.
Storlazzi, E. D., and Elkins, H. B. (1941). *J. industr. Hyg.*, **23**, 459.
Swan, A. A. B. (1960). Personal communication.
Totušek, J., and Waelschová, A. (1954). *Pracov. Lék.*, **6**, 99.
Turrian, H., Grandjean, E., and Turrian, V. (1956). *Schweiz. med. Wschr.*, **86**, 1091.
Wirth, W. (1936). *Naunyn-Schmiedeberg's Arch. exp. Path. Pharmak.*, **184**, 91.

Brit. J. industr. Med., 1961, **18**, 295.

THE TOXICITY OF PRECIPITATED SILICA

BY

P. D. BYERS and J. C. GAGE

From the Postgraduate Medical School, Hammersmith and Imperial Chemical Industries Limited, Industrial Hygiene Research Laboratories

(RECEIVED FOR PUBLICATION APRIL 4, 1961)

The proportion of respirable particles in dust clouds generated from three samples of precipitated silica has been shown to range between one-quarter and one-third by weight.

After a single intratracheal dose of the silicas to rats, chemical analysis shows a progressive disappearance of silica from the lungs, though it is still detectable after 12 months. Some silica appears in the liver and kidneys but in two of the three samples none remains after 12 months.

The nature and duration of the lung lesions produced in rats after a single intratracheal injection are described. A mild degree of fibrosis was observed which showed a steady regression with time and was to some extent influenced by the nature of the silica injected. The lesions showed little resemblance to those arising from quartz and were more akin to those produced by non-fibrogenic dusts.

Recommendations are made for the precautions to be taken during the industrial handling of these dusts.

Amorphous silica has been used for many years in a variety of industries, notably for inclusion into rubber formulations as a filler. Although it appears to have been handled without special precautions, no authenticated case of silicosis or other respiratory disease has been traced which can be attributed to this agent. Nevertheless, the increasing industrial usage of this material has aroused some concern lest hitherto unsuspected toxic properties might emerge.

Amorphous silica is not a clearly defined chemical entity and several methods are employed for its commercial production. Most of the experimental investigations into the toxicity of amorphous silica have been concerned with "flame" silica, prepared by burning a mixture of hydrogen and silicon tetrachloride vapour. It has been shown that this type of silica has a cytotoxic action (Policard and Collet, 1957) and can be lethal when injected intravenously (Swensson, Glomme, and Bloom, 1956), but that when inhaled it produces a reversible lesion in the lungs with only a mild degree of fibrosis and with very little collagen deposition (Schepers, Delahant, Bailey, Gockeler, and Gay, 1957a). Similar effects on the lungs have been shown by intratracheal injections (Klosterkötter and Jötten, 1953).

Another commercially important type of amorphous silica is prepared by acid precipitation from an aqueous solution of sodium silicate, a process similar to that used for the preparation of silica gel. There do not appear to have been any systematic investigations into the toxicity of this precipitated silica, which cannot be assumed to be identical with that of "flame" silica as there is no certainty that the two substances have the same composition. An investigation of precipitated silica from two manufacturers has, therefore, been undertaken, in order to assess the possibility of a silicosis hazard from the industrial use of this material.

Fibrogenic properties of a dust can be determined by studying the cellular reaction to the dust within the lung. Experimentally, dust can be introduced by exposing animals to a dusty atmosphere or injecting dust in some form of suspension directly into the lung. In the former method, the animals automatically select those particles which can enter the respiratory system sufficiently far to be retained (Dautrebande, Beckmann, and Walkenhorst, 1957). In the injection method, the size distribution of the material depends on the degree of dispersion in the suspension administered. Apart from considerations of particle size, the injection method probably provides at least as severe a test of the fibrogenic capacity of a dust as do dusting procedures (Schepers, Durkan, Delahant, Creedon, and Redlin, 1957b; Belt and King, 1945; Kettle and Hilton, 1932).

Amorphous silicas are composed of very fine particles, the average diameter being in the region of 20 mμ, which tend to aggregate loosely in air. The size of these aggregates will determine the respirable fraction of the dust, but will not necessarily determine the effect of the dust once it has entered the body, since the silica may then be exposed to other influences causing a variation in the degree of aggregation.

We have performed two types of experiment with three amorphous silicas. In the first, the proportion of respirable particles in air-borne precipitated silica dust clouds has been measured. In the second we have administered to rats, by intratracheal injection, single doses of the samples, and have studied the distribution and excretion of silica and the nature and duration of the lesions produced.

Samples Investigated

Samples of precipitated silica were obtained from two manufacturers. From one supplier two samples were received; sample A1, the quality normally supplied, and the sample A2 which had the same characteristics when originally manufactured, but which had for reasons not clearly known increased in particle size on ageing. Sample B was obtained from the second supplier.

Sample A1.—The particle size was 19 mμ, calculated by an air permeability method (Pechukas and Gage, 1946).

Sample A2.—When freshly prepared this was shown by electron microscopy to have a particle size of 20 mμ and a specific surface by gas absorption of 110 $m.^2/g.$ A later measurement of the specific surface showed that this had decreased to 60 $m.^2/g.$ and an air permeability measurement gave a particle size of 60 mμ.

Sample B.—The particle size by an air permeability method was 25 mμ.

The intravenous toxicity of the three samples has been determined on rats. Most deaths occurred within two hours of injection and all rats surviving for 24 hours subsequently recovered. The following LD_{50} values were obtained, with the fiducial limits ($p = 0.05$) given in parentheses: sample A1, 35·2 (32-39); sample A2, 41·2 (34·5-42); sample B, 44·4 (40·5-49) mg./kg. These figures are appreciably higher than the values 7-17 mg./kg. obtained on mice with "flame" silica by Swensson *et al.* (1956).

The Respirable Fraction of Precipitated Silica

Theoretical and experimental studies have shown that the ability of dust particles to penetrate into the lungs when inhaled is related to their free falling speed in air and is, therefore, determined by their size and density (Davies, 1952). It is thought that quartz particles with a diameter greater than about 7 μ are trapped in the upper respiratory passages and do not enter the lung alveoli and give rise to silicosis (Wright, 1954). The ultimate particle size of precipitated silica is very much less than this figure, but the particles are associated into loose aggregates whose density will be much lower than that of quartz. The upper size limit for penetration into the lung by these aggregates is uncertain but will be greater than the 7μ quoted for quartz. A classification of a precipitated silica dust cloud by direct measurement of aggregate size would, therefore, have only a limited value as a guide to the pneumoconiosis hazard from the dust, and it would be preferable to base the size analysis on the free falling speed of the particles. A determination of the aggregate size range by trapping the dust in a liquid followed by elutriation would probably be unsuitable as the forces at the solid-liquid interface might change the degree of dispersion. The instrument selected for studying the precipitated silica dust was the Hexhlet Air Elutriator (Casella Ltd.) which collects the fraction of the dust with a free falling speed in air sufficiently low to permit penetration into the lungs (Wright, 1954).

Apparatus

The dust cloud was generated by a modification of the United States Bureau of Mines Apparatus (Schrenk, 1939), in which the dust is filled into a long vertical cylindrical container which is slowly raised at a uniform speed to maintain the surface of the dust in contact with a static head. From this head the dust is aspirated into the air stream by means of a Venturi jet. The original apparatus was designed for a much larger scale than was convenient for these experiments and an attempt to reduce it in size was unsatisfactory as an intermittent dust feed was obtained. This difficulty was overcome by applying an oscillatory rotation to the dust container and a positive flow control to the air supply used to pick up the dust from the static head and introduce it into the main air stream. The apparatus used is shown diagrammatically in Fig. 1. The dust container, A, was constructed from a precision 5 mm. bore glass tube, 20 cm. long, surmounted by a B 14 standard taper socket. A side arm, B, was connected through a flow meter to an air line with flow control. A brass piston, C, 12 mm. long and bearing 12 longitudinal flutes, slid easily into the tube A. A thin-walled stainless steel tube, D, 1·5 mm. diameter and 25 cm. long was fixed into a hole bored centrally in the piston. This tube also passed through a hole drilled in a "Perspex" cone, E, fitted in the B 14 socket and forming an air-tight seal but permitting movement of the tube in the cone. The upper end of the tube was clamped and the tip entered a hole in the base of an open-ended "Perspex" box attached to the end of the Hexhlet apparatus.

The dust container was mounted in a bored rubber stopper in an adaptor on the ram, F, of a slow injection apparatus (C. F. Palmer Ltd.) which had been modified by the makers for a vertical drive. This apparatus was capable of raising the dust container a distance of 4 in. at constant rates which could be varied between 1 in. in 10 to 320 minutes. A horizontal slotted bar, G, was also attached to the rotating head of the ram and in the slot was a vertical pin, H, on an adjustable crank which turned by means of a geared down electric motor at a speed of 30 r.p.m. The vertical pin on the crank was sufficiently long to impart an oscillating angular motion of about 65° to the slotted bar, whatever the position of the ram.

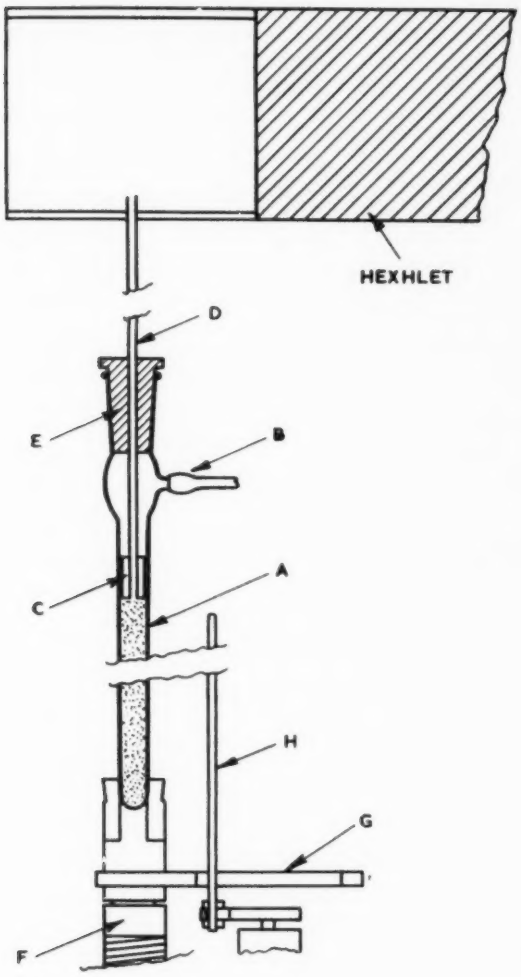


FIG. 1.—Apparatus for the generation of silica dust clouds. A, dust container; B, entry of air for dust pick-up; C, fluted brass piston; D, stainless steel tube; E, "Perspex" cone; F, ram of slow injection apparatus; G, slotted bar; H, rotating pin.

A weighed amount of the silica sample to be examined was introduced in portions into the dust container, the tube being tapped between each addition. The apparatus was assembled as shown in Fig. 1, and with a soxhlet thimble of known ash weight in the Hexhlet apparatus. The air line to the Hexhlet was turned on, and 0·2 l./min. dry air was passed through the side arm of the dust reservoir. The vertical drive on the dust container was started at a speed of 1 in. in 40 min. and the agitator switched on. By this means the dust was caused to issue at a known rate from the tip of the vertical tube and enter the air stream passing at a rate of 100 l./min. into the Hexhlet.

The run on each sample was continued for two to three hours at the end of which the dust remaining in the container was weighed and hence the total injected into the air stream was calculated. The soxhlet thimble was removed and ashed and the residue weighed and corrected for the ash of the thimble and the measured percentage of loss in weight of the silica sample at the ashing temperature. The results of duplicate experiments on the three samples are shown in Table 1.

The figures in the third column of Table 1 show that the respirable fraction of the precipitated silica samples ranged between 25 and 40%. Both samples from manufacturer A showed a smaller proportion of fine particles than the sample B. The apparently greater aggregate size of sample A2 may be related to the statement that this sample had changed in its physical properties on ageing leading to an increase in the ultimate particle size, and also to the observation made in subsequent experiments that this sample is more difficult to disperse in water. The variation in the atmospheric concentration in duplicate experiments shown in Table 1 must be attributed to differences in the degree of packing of the powder in the dust container; nevertheless, the duplicates on the percentage reaching the filter are in tolerably good agreement.

TABLE 1

RESPIRABLE FRACTION OF PRECIPITATED SILICA DUST

Sample	Weight Silica (mg.)		% Silica on Filter	Duration (min.)	Atmospheric Concentration (mg./m.^{3})
	Injected	Collected			
A1	369	107	29·0	130	28·4
	285	96	33·7	120	23·7
A2	603	148	24·5	170	35·5
	479	129	27·0	170	28·2
B	457	198	43·3	170	26·9
	280	98	35·0	120	23·3

Retention of Precipitated Silica in Lungs

Groups of 50 male and 50 female adult Wistar strain albino rats were injected intratracheally under light ether anaesthesia with 1 ml. of a 2·5% suspension of each of the samples of precipitated silica. The silica was sterilized in hot air at 160° C. for two hours and dispersed in sterile isotonic saline by means of a Griffith's tube. Higher doses than 25 mg./rat caused death within 24 hours. The first injections were made with sample A2 and although initially all rats tolerated the dose well, 12 of the group died within four days with post-mortem signs of consolidation and abscess formation in the lungs. In experiments on the other two samples, which were started several months later, 2,000 units of penicillin were included in the injection and no immediate deaths from lung infection occurred. At three intervals during the 12 months after injection five male and five female rats were killed and the lungs, liver, kidneys, and spleen were taken for silica determinations. Similar groups were killed for a histopathological examination of the lungs.

Silica was determined in the tissues by a modification of the method of King, Stacy, Holt, Yates, and Pickles (1955). A 50% aqueous homogenate of each sample was prepared and 2 g. was fused with 2 g. sodium carbonate in a platinum crucible. Heating was gentle during charring of the tissue and was then applied more strongly until the melt remained white on cooling. The melt was dissolved in water by gently warming and was then cautiously neutralized to Congo Red with 10 N sulphuric acid, and a further 0·2 ml. of the acid added. The solution was diluted to 50 ml. with water and 1 or 2 ml. portions taken for analysis. The subsequent colorimetric determination of silica followed the method described by King *et al.* (1955), the optical density of the blue colour being measured at 810 mμ. The results of these analyses, expressed as μg. SiO_2 per rat, are given in Table 2, together with the average silica content of tissue from normal rats.

TABLE 2

RETENTION OF SILICA BY RAT TISSUES

Sample	Weeks After Injection	Lungs	Liver	Kidneys	Spleen
A1	12	1,570	177	33	0
	24	755	20	12·4	1·4
	52	210	61	9·7	0
A2	12	5,150	249	138	0
	36	1,550	153	44	19
	52	720	153	56	0
B	12	652	234	34	5·8
	24	324	43	8·9	1·4
	52	108	38	12	0
Average silica content of normal rat tissue		28	37	11	5

All rats received an intratracheal dose of 25 mg. precipitated silica. Figures represent μg. SiO_2 per rat.

Effect of Precipitated Silica on Lungs

Groups of five male and five female rats, dosed intratracheally with each of the precipitated silicas as described in a previous section, were taken at three intervals over a period of 12 months for histopathological examination of the lungs. Sections of the whole lungs, cut in the frontal plane, were stained with haematoxylin and eosin, and others impregnated with silver for reticulin fibres. Other sections were micro-incinerated, with and without acid-washing, and a few were stained with Van Giesen.

An examination of the acid-washed incinerated sections showed that the distribution of silica in the lungs was not uniform, but occurred in small areas throughout one or both lungs with occasionally a large amount deposited in one lung segment. The stained sections showed that the three dusts elicited the same type of response in the lungs and that there was no significant difference between the effects produced in male and female rats. The silica particles were contained within macrophages which were aggregated into foci around terminal and respiratory bronchioles. Small numbers of lymphocytes and fibroblasts surrounded these foci and were also intermingled within the macrophages (Figs. 2 and 3), while reticulin fibres were woven through and around the lesion. These general features of the dust lesions were to some extent influenced by the distribution of dust within the lung. Where dust deposition was very heavy the structure of the lung segments was completely obliterated by the presence of the lesions produced. In these areas the basic lesions consisted as before of a group of macrophages with a few fibroblasts and lymphocytes, but they were contiguous and their individuality was only distinguishable by virtue of the peripheral zone of fibroblasts and lymphocytes (Fig. 3). These confluent lesions were maximal 12 weeks after injection; with the passage of time dust was gradually eliminated from them, with some contraction of the lesion resulting in varying degrees of deformity of the lung. The individual lesions also decreased in size and number with time.

The size and number of the lesions was a function of the quantity of dust present in the lungs. For a given dust the lungs with the larger amounts of dust had the larger lesions and with the progressive elimination of the silica from the lungs there was a corresponding decrease in the size of the lesions and, towards the end of the experiment, in their number. The maximal size of the lesions was seen in the first groups of animals examined, 12 weeks after injection. In these sections the reticulin fibres were also most numerous and the amount tended to decrease as the lesions decreased in size during the course of the experiment.

There was little evidence that the lymphatic system was involved in the process of dust elimination. Within the lung itself dust was not often seen in lymphoid tissue. In those sections where paratracheal lymph nodes were present none or only small quantities of dust were seen. The small quantity which could be detected was contained within macrophages lying in sinusoids, and elicited virtually no reaction (Fig. 5). These observations do not preclude the possibility that significant quantities of dust were eliminated through the lymphatics.

The type of dust used affected the nature of the lesion, in the main by virtue of the different rates of excretion of the three samples; inspection of the micro-incinerated sections confirmed the analytical evidence that sample B was the most quickly eliminated from the lung and sample A2 the slowest. There appeared to be slight differences between the dusts in addition to this; sample A2 tended to have slightly

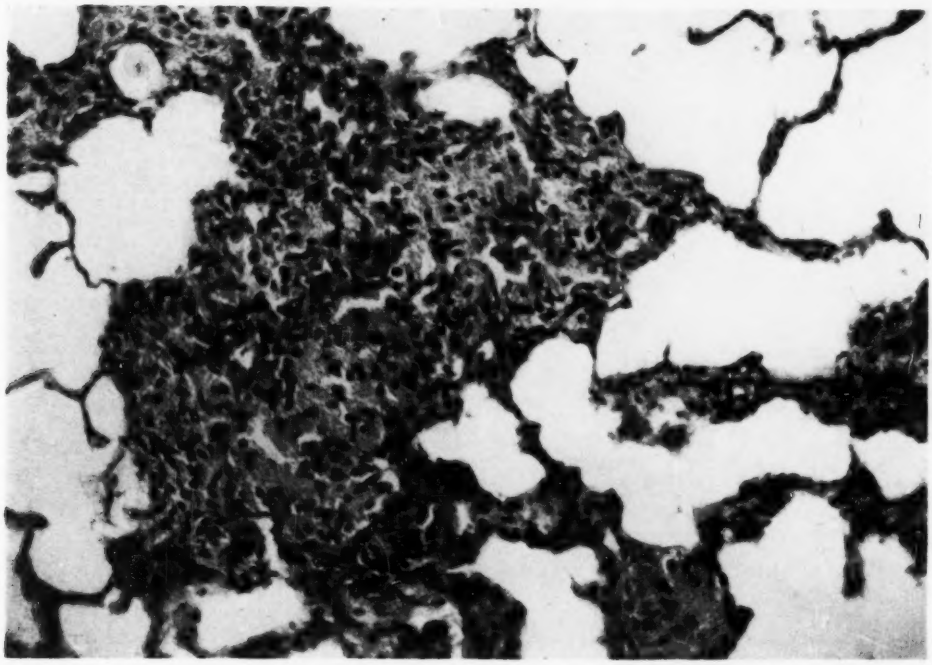


FIG. 2.—Rat lung 12 weeks after an intratracheal injection of 25 mg. of amorphous silica (sample B): an isolated lesion composed principally of macrophages. (H. and E. × 188.)

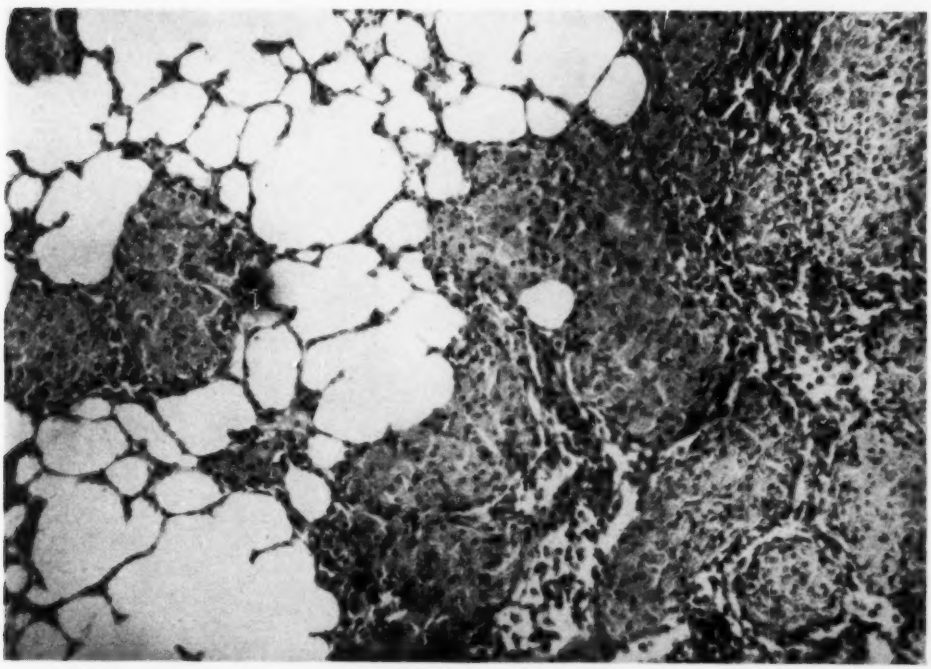


FIG. 3.—Rat lung 12 weeks after an intratracheal injection of 25 mg. of amorphous silica (sample A1): an isolated lesion at the left and confluent lesions to the right. (H. and E. × 100.)

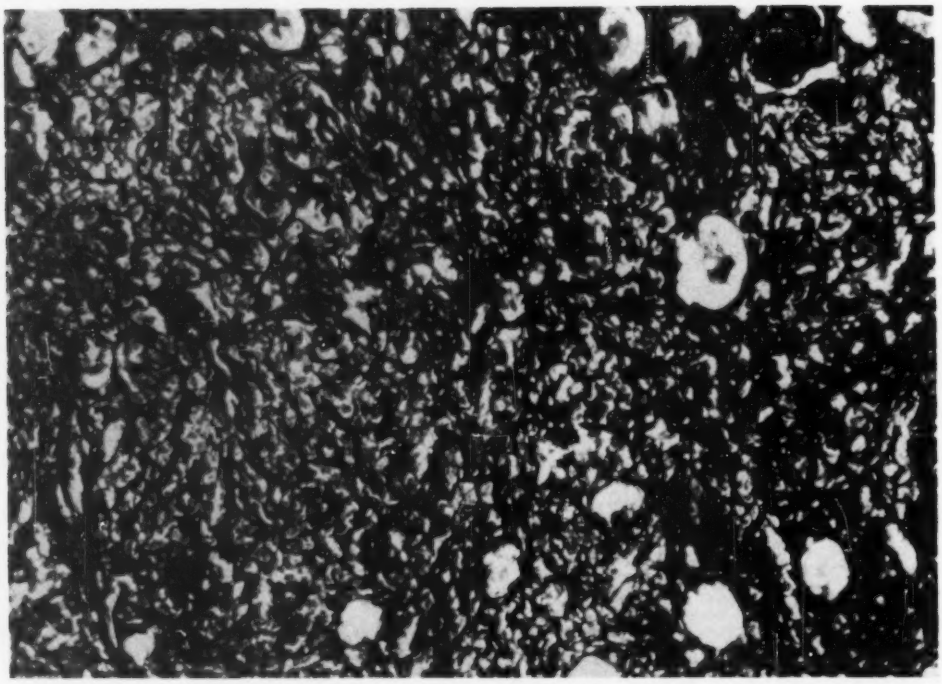


FIG. 4.—Rat lung 52 weeks after an intratracheal injection of 25 mg. of amorphous silica (sample A2): a portion of a confluent lesion showing loss of lung architecture and proliferation of reticulin fibres, with some collagen fibres. (Silver reticulin, neutral red × 150.)

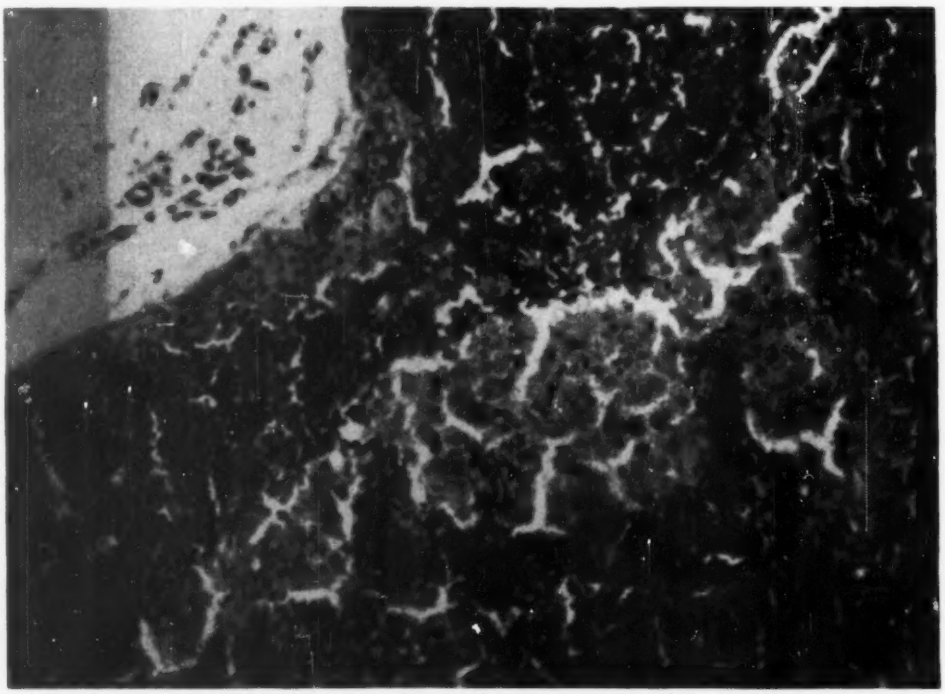


FIG. 5.—Rat mediastinal lymph node (the same animal as Fig. 4), 52 weeks after an intratracheal injection of 25 mg. of amorphous silica (sample A2): sinusoidal collections of macrophages with no associated inflammatory response or fibroblastic proliferation. (H. and E. × 200.)

larger lesions than the others for the same amount of dust, and also to induce a greater amount of fibroblastic proliferation which had the appearance of being more mature. As the lesions from this dust became smaller with time there was some condensation of the reticulin fibres giving rise to a rather more dense distribution at the periphery. In the first batch of animals examined, these lesions showed grade 1, or in one animal grade 2, fibrosis by Belt and King's (1945) classification, and at the termination of the experiment the lesions were graded as 1 minimal to 2 (Fig. 4).

The rats injected with sample A1 showed a slow and steady regression of the isolated lesions with an accompanying reduction of the reticulin fibres and no evidence of their maturation. The first batch examined showed lesions with grade 1 maximal to 1 fibrosis and at the end of the experiments the grades were 1 to 1 minimal; at this point sections from five animals showed no isolated lesions, while in others they were few and small. In areas of heavy dust deposition a rather milder effect was elicited than that from sample A2, and regression went further over the period of observation. The lesions from sample B were initially similar to those from samples A1 (Figs. 2 and 3), but their rate of regression was more rapid and the final sections showed either a complete resolution or small scattered dust foci with few reticulin fibres around or within the lesion (fibrosis grade 1 to 1 minimal). There also remained a few areas of confluent lesions which were small and irregular and showed a rather light reticulin network and a little retraction.

In general the effect of the dust on lung function appeared to be small. There was evidence of mild emphysema in a few lungs but apart from a few large abscesses and foci of bronchiectasis there was little evidence of infection. Pneumonia and bronchopneumonia were infrequent. It does not seem likely that the presence of dust in the lungs greatly influenced the incidence or course of these infections, an opinion that tended to be confirmed by examination of 10 normal adult male and female rat lungs. In these animals microscopic and macroscopic inflammatory lesions were present with a similar overall frequency as in the lungs of the experimental animals.

Conclusions

The presence of particles of respirable size, to the extent of between one-quarter and one-third by weight, in dust clouds generated from the three samples of precipitated silica is evidence that the extent of aggregation is not sufficiently great to cause the dust to be entirely retained in the upper respiratory passages when inhaled and so preclude any cellular reaction in the lungs.

When precipitated silica is injected into rats' lungs a significant amount can still be detected by chemical analysis after a period of 12 months. The rate of elimination is, however, much faster than that observed with quartz and other fibrogenic dusts. Initially some of the silica leaving the lung appeared in the liver and kidneys, but with samples A1 and B no significant amount of silica could be detected in these organs six months after injection. Sample A2 was more persistent and was found in the liver and kidneys 12 months after injection. There is no evidence to indicate the route by which silica is eliminated from the lungs, and whether it is transported in the body as particles or in solution.

The lesions produced by the intratracheal injection of precipitated silica show little resemblance to those arising from quartz and were more akin to the effect of non-fibrogenic dusts. The most marked effect on the lungs was produced by sample A2 where there was a tendency for the lesions to coalesce with a loss of well-defined boundaries, but this in no way resembled the progressive reaction to quartz which produces an expanding lesion with maturation of fibres. Another notable difference resided in the evident regression of lesions during the course of the experiment, corresponding to the disappearance of silica from the lungs.

The severity of the lesions from precipitated silica was to some extent affected by the nature of the dust. Sample A2 produced the largest and most persistent lesions and also a slightly greater deposition of fibres. It seems unlikely that any chemical difference existed between samples A1 and A2, which were initially made by identical processes, and it seems more probable that the greater activity of sample A2 can be attributed to those surface properties which resulted in its greater tendency to aggregate. This tendency could be observed both from the small proportion of fine particles in a dust cloud from this sample, and in the greater difficulty which was encountered in preparing an aqueous dispersion from it.

The single intratracheal dose technique is regarded as an efficient means of detecting the fibrogenic properties of a dust, and the absence of such a cellular reaction when precipitated silica is tested in this manner suggests that silicosis is unlikely to be encountered when materials of this type are used in industry. This does not imply that they are completely innocuous and precautions common to all other dusty materials should be taken to keep atmospheric contamination to a minimum. Where this is impossible, it is desirable that approved respiratory protection should be worn. Workmen exposed to these dusts should be subjected to periodic medical supervision, and should not be allowed to continue

in their employment if there are signs of chronic respiratory disease.

Technical assistance in this investigation was provided by Mr. R. T. Ashmole and Mr. P. Salt.

References

Belt, T. H., and King, E. J. (1945). *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 250. H.M.S.O., London.
Dautrebande, L., Beckmann, H., and Walkenhorst, W. (1957). *Arch. industr. Hlth*, **16**, 179.
Davies, C. N. (1952). *Brit. J. industr. Med.*, **9**, 120.
Kettle, E. H., and Hilton, R. (1932). *Lancet*, **1**, 1190.
King, E. J., Stacy, B. D., Holt, P. F., Yates, D. M., and Pickles, D. (1955). *Analyst*, **80**, 441.
Klosterkötter, W., and Jötten, K. W. (1953). *Arch. Hyg. (Berl.)*, **137**, 625.
Pechukas, A., and Gage, F. W. (1946). *Industr. Engng Chem. (Anal. ed.)*, **18**, 370.
Policard, A., and Collet, A. (1957). *Arch. Mal. prof.*, **18**, 50.
Schepers, G. W. H., Delahant, A. B., Bailey, D. A., Gockeler, E. L., and Gay, W. C. (1957a). *Arch. industr. Hlth*, **16**, 499.
——, Durkan, T. M., Delahant, A. B., Creedon, F. T., and Redlin, A. J. (1957b). *Ibid*, **16**, 125.
Schrenk, H. H. (1939). *U.S. Bureau Mines I.C.* 7086.
Swensson, A., Glomme, J., and Bloom, G. (1956). *A.M.A. Arch. industr. Hlth*, **14**, 482.
Wright, B. M. (1954). *Brit. J. industr. Med.*, **11**, 284.

THE JULY (1961) ISSUE

The July (1961) issue contains the following papers:—

The Effect of Film Quality on Reading Radiographs of Simple Pneumoconiosis in a trial of *X*-ray sets. By F. D. K. Liddell.

A Survey of the Methods Developed in the National Coal Board's Pneumoconiosis Field Research for Correlating Environmental Exposure with Medical Condition. By J. W. J. Fay and J. R. Ashford.

Sickness Absence of Women Bus Conductors in London Transport (1953-1957). By C. J. Cornwall and P. A. B. Raffle.

Olfaction Tests. By I. Dingwall Fordyce.

Anosmia in Alkaline Battery Workers. By R. G. Adams and Norman Crabtree.

The Kidney in Lead Poisoning. By Zdenko Radošević, Marko Šarić, Tihomil Beritić, and Jelica Knežević.

Trithion Poisoning. By C. E. D. Hearn.

A Study of 104 Cases of Migraine. By A. J. Childs and M. T. Sweetnam.

Book Reviews

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 17*s*. 6*d*.

Brit. J. industr. Med., 1961, **18**, 303.

POISONING BY ETHYL MERCURY TOLUENE SULPHONANILIDE

BY

M. A. JALILI and A. H. ABBASI

From the Department of Medicine, College of Medicine, University of Baghdad, Iraq

(RECEIVED FOR PUBLICATION JULY 2, 1961)

Poisoning by a fungicide used for seed-borne diseases of cereals, ethyl mercury *p*-toluene sulphonanilide (Granoson 1, Dupont), is described. It affected a large number of farmers and their families who used the dressed seed in the preparation of home-made bread. Many systems were involved, including the kidneys, the gastro-intestinal tract, the skin, the heart, and the muscles, but involvement of the nervous system was the most constant with disturbance of speech, cerebellar ataxia, and spasticity. Mental abnormalities were occasionally observed. Many patients died. The changes in the electrocardiograms are described and illustrated. The importance of effective measures for prevention and the need for further studies of treatment are stressed.

Mercury poisoning is nowadays comparatively rare, and when seen is generally the result of the medical use of, or accidental exposure to, the metal or its salts. However, when organic mercury compounds are used as fungicides against seed-borne diseases of cereals and are handled by a large number of farmers who are not used to such toxic materials, mercury poisoning is likely to occur.

Organic mercury compounds have been used to prevent seed-borne diseases of cereals since 1914. Many different preparations of mercury are sold as fungicides and typical examples used widely are phenyl mercury acetate, ethyl mercury phosphate, and methyl mercury dicyandiamide. In the past few years ethyl mercury *p*-toluene sulphonanilide (Granosan M, Dupont) has been used in Iraq as a fungicide for seed wheat, and hundreds of cases of poisoning from this material occurred. The present communication deals with the clinical picture of the disease and the electrocardiographic changes.

Incidence

In 1956 many cases of mercury poisoning were observed in the North of Iraq, and more than 100 cases were admitted to Mosul Hospital with 14 deaths. In 1960, many farmers from the central part of Iraq were affected and 221 patients (106 males and 115 females) were admitted to one hospital in Baghdad. Other patients went into other hospitals. There were many deaths, and patients usually spoke about deaths in their own families and locality. According to Dr. W. M. Ali (personal communication) 22 bodies were examined in the Medico-Legal Institute in Baghdad, and large amounts of mercury were found in the organs. For example, in the liver the level was 6·58 ± 1·85 mg./100 g.

Patients admitted that they were warned against eating the wheat because it had been treated with a harmful material. Some of them washed it to rid it of the poison; and when they noticed that nothing happened to fowls that consumed it for a few days, they started eating it, sometimes mixed with larger amounts of untreated wheat or maize. People who had eaten the dressed wheat and remained well for some days or weeks, set a bad example to others who did not hesitate to consume the dressed wheat in the form of bread.

Method and Materials

Granosan M (Dupont) was mixed with seed wheat, 50 g./100 kg. This commercial product contains 7·7% ethyl mercury *p*-toluene sulphonanilide. Its total mercury content, as mercury metal, is 3·2%. In October, November, and December 1955, the dressed seed was distributed to farmers in the North of Iraq and cases of poisoning appeared in January, February, March, and April 1956. One of us (M.A.J.) examined many such cases at Mosul Hospital. In the season of 1959-60 the dressed seed was distributed in the central part of Iraq, and many patients were admitted to Baghdad hospital in March and April, and to a lesser extent in the following months.

The present series consists of 26 cases examined by the

authors. The patients were farmers or their wives and children from the rural areas of different parts of Iraq. Usually many members of the family were affected. Their ages ranged between 7 and 60 years with an average of 23 ± 12 years. One case occurred among the workers who dressed the wheat. In addition to the case history and clinical examination, the urine was examined; 12-lead electrocardiograms were taken in 15 patients.

The following case records illustrate the condition.

Case Reports

Case A.—A landworker, aged 17, purchased the treated wheat from fellow farmers and mixed it with ordinary wheat. Ten days after eating the bread made from it symptoms began to appear. Most of his diet consisted of home-prepared bread. At first he experienced severe pain in the knees; 10 days later he had generalized deep-seated pains; three weeks later he developed pruritus in the soles, palms, and genitalia, and his vision was affected. Bowels moved once or twice daily; there was some oliguria and little cough with mucoid expectoration. He was admitted to hospital two months after the onset of symptoms. Examination revealed a thin patient, suffering from pain. There was a grey zone on two lower incisors, coarse twitchings in the limb muscles, muscular excitability on percussion of the muscles, tender muscles but no tenderness along the course of nerves; there was no oedema, jaundice, or anaemia. Heart, chest, and abdomen showed no clinical abnormality. Tendon reflexes were present and the plantars were flexor. There was no ataxia or nystagmus. Fundi showed no lesion at this stage. Urine contained a trace of albumin and uric acid crystals. Electrocardiogram was normal.

Case B.—A farmer aged 16, ate bread made from treated seed two months before admission. After six weeks he developed abdominal pain, headache, weakness of the legs, difficulty in walking, unsteadiness when standing, and progressive deterioration of vision. This was accompanied by polyuria and polydipsia. Two other brothers were similarly affected. On admission he was thin, with clear mentality, almost blind, and had slurred speech with marked ataxia. There was a little pallor of the temporal parts of the optic discs. There was some spasticity of the lower limbs, and the tendon reflexes were diminished. Plantar reflexes were flexor and abdominal reflexes absent. Pulse was 100/min. irregular; blood pressure 130/70 mm. Hg, and temperature 37°C. No cardiac murmurs, or added sounds in the chest; liver and spleen were not palpable; there were cautery marks on the abdomen (in rural areas of Iraq cautery is often made for pain). Urine showed a trace of albumin and granular casts. Blood urea 45 mg./100 ml., Hb 70% (Sahli), red blood cells numbered 3,800,000, and white blood cells 9,800. Electrocardiogram showed a shifting pace-maker.

The patient was treated with BAL and after five days his ataxia and vision markedly improved.

Results and Discussion

Table 1 gives the main symptoms and signs found in the 26 patients examined by the authors. It is evident that they differ from the symptoms and signs of poisoning by mercury or its inorganic salts. Mercury poisoning has followed application of ointments in isolated cases (Stoneman, 1958; Williams and Bridge, 1958). Infants have been poisoned from a calomel used in teething preparations (Wilson, Thomson, and Holzel, 1952). Ingestion of mercuric bichloride leads to two kinds of lesions: those of the gastro-intestinal tract due to its corrosive action and those due to the specific kidney involvement (Troen, Kaufman, and Katz, 1951). On the other hand the clinical picture of poisoning with organic mercurial compounds is said to be confined to damage of the central nervous system and consists of deafness, generalized ataxia, dysarthria, progressive visual deterioration, dysphagia, sphincteric incontinence, mental confusion, stupor, and death (Locket, 1957).

Hunter, Bomford, and Russell (1940) described four cases of poisoning by inhalation of methyl mercury compounds in a factory. In these cases, with the exception of tremor, symptoms of poisoning by metallic mercury, namely salivation, stomatitis, and erethism, were absent, and the nervous system alone was involved, with severe generalized ataxia, dysarthria, and gross constriction of the visual fields. In experiments on rats and a monkey, methyl mercury compounds caused a widespread degeneration of certain sensory paths of the nervous system; the peripheral nerves and the posterior spinal roots were affected first, and the spinal cord and certain neurones of the middle lobe of the cerebellum later. Acute degeneration and sometimes early fibrosis of the kidneys were found in these rats. Though much of the acute damage in man apparently subsides, necropsy suggests that localized cerebellar degeneration does occur (Hunter and Russell, 1954). Lundgren and Swensson (1949) have also described cases of poisoning by methyl mercury derivatives used as fungicides on grain.

The present series represents the severe cases of poisoning admitted to hospital. The clinical picture found in the cases seen in Mosul (northern Iraq) in 1956 and those seen in Baghdad in 1960, falls into the following groups, depending on the amount of the poison taken and the system or systems mainly involved:

1. *Involvement of the kidneys.*—Oliguria may develop at the beginning of the illness if large amounts are consumed. Oedema is rarely seen. Usually polyuria and polydypsia with loss of weight and sometimes dehydration develop. These were the main symptoms in 62 cases out of 86 in the north of Iraq (R. Tapponi, personal communication). In Tapponi's cases urine examination showed the

TABLE 1

MAIN SYMPTOMS AND SIGNS

Case No.	Age (years)	Symptoms															
		Duration (days)	Polyuria	Vomiting	Bowels	Abdominal Pain	Skeletal Pain	Inability to Walk	Ataxia	Slurred Speech	Impaired Vision	Tremor	Twitching	Mentality	Pruritus	Tendon Reflexes	Abdominal Reflexes	Plantar Reflexes
1	25	90	+	+	Diarrhoea	−	−	+	+	+	−	−	−	Normal	+	+ +	+	Flexor
2	7	7	+	+	−	+	−	+	Drowsy	Drowsy	?	−	−	Drowsy	−	+ + +	+	Extensor
3	17	5	+	−	−	−	+	+	+	+	+	−	−	Normal	−	+ + +	+	Extensor
4	22	7	+	+	Diarrhoea	+	−	+	−	Aphasia	?	−	−	Normal	−	+ + +	+	Extensor
5	14	14	+	+	Diarrhoea	+	−	+	−	Aphasia	?	−	−	Excitable	−	+	+	Extensor
6	35	10	−	−	−	+	−	+	+	+	−	−	−	Normal	−	+	+	Extensor
7	16	10	−	+	Diarrhoea	−	−	+	−	−	−	−	−	Normal	−	+ + +	−	Extensor
8	10	40	−	−	−	+	−	+	−	Aphasia	−	−	−	Normal	−	+	+	Flexor
9	16	12	+	−	−	−	−	+	+	+	−	−	−	Normal	−	+ + +	−	Extensor
10	23	7	−	+	Diarrhoea	+	+	+	+	Aphasia	?	−	−	Psychotic	−	+ + +	+	Extensor
11	8	31	+	−	−	+	−	+	+	+	+	−	−	Normal	−	+ + +	−	Extensor
12	25	32	+	−	−	+	−	+	+	+	+	−	−	Normal	−	+ + +	−	Extensor
13	30	60	+	−	−	−	+	−	−	−	−	+	−	Normal	+	−	+	Flexor
14	9	10	+	+	Constipation	−	−	+	+	+	+	+	−	Normal	−	+ + +	+	Flexor
15	10	15	−	−	−	−	−	+	+	+	+	−	−	Normal	−	+	+	Flexor
16	16	15	+	−	−	+	−	+	+	+	+	−	−	Normal	−	−	−	Flexor
17	16	60	+	−	Diarrhoea	+	+	+	+	+	−	−	−	Normal	−	+ +	+	Flexor
18	35	15	−	+	Diarrhoea	+	−	+	+	+	?	−	−	Euphoria	−	+	+	Flexor
19	35	8	−	−	−	−	−	−	+	−	−	+	−	Psychotic	−	+ +	+	Extensor
20	40	120	−	−	−	−	+	+	−	−	−	−	+ +	Normal	+	+	+	Flexor
21	12	30	+	−	−	−	−	−	+	−	−	−	−	Normal	+	+	+	Flexor
22	35	15	−	+	Constipation	+	+	+	−	−	−	−	+ +	Normal	+	+ +	+	Flexor
23	17	60	Oliguria	−	−	−	+	−	−	−	+	−	+	Normal	+	+	+	Flexor
24	20	90	−	−	Constipation	+	+	+	−	−	−	−	+	Normal	−	+	+	Flexor
25	26	25	Oliguria	−	Constipation	+	+	−	−	−	+	−	+	Normal	−	+	+	Flexor
26	35	60	+	−	Constipation	+	+	−	−	−	−	−	+	Normal	−	+ + +	+	Flexor

Sequence of events and other findings:

Case 1: Dark pigmentation of skin and mucous membranes, with exfoliation of skin; ataxia, paraesthesiae; then polyuria and polydipsia. *Case* 2: Vomiting, abdominal pain, marked weakness of legs with paraesthesiae, spastic paraplegia. *Case* 3: After eating dressed seeds for one month developed severe polydipsia and polyuria with pain in legs and inability to walk. Spastic paraplegia, optic atrophy. *Case* 4: Vomiting, colic, diarrhoea, inability to speak or walk. *Case* 5: Abdominal colic, tarry stools, vomiting, asthenia, polyuria and polydipsia, incontinence of urine. Severely ill, with opisthotonos, severe excitement. *Case* 6: Abdominal pain; excessive salivation, weak legs, general spasticity, dysarthria. *Case* 7: Weakness of legs, vomiting, general spasticity. *Case* 8: Fever, abdominal pain, gradual aphasia and dysphagia, slight weakness of legs. *Case* 9: Gradual weakness of limbs, incontinence of urine, dysarthria; quadriplegia. *Case* 10: Vomiting, diarrhoea, abdominal colic, inability to walk or speak. *Case* 11: Abdominal pain, burning micturition, paraesthesiae, spastic paraplegia and slurred speech. *Case* 12: Headache, abdominal colic, burning micturition, paraesthesiae, slurred speech, spastic paraplegia. *Case* 13: Severe pain in hips and legs not responding to usual analgesics. Paraesthesiae, tremors; pruritus and exfoliation of legs and feet. *Case* 14: Polydipsia, vomiting, constipation, numbness, weakness of legs, unsteady gait, slight impairment of hearing, then blindness; spasticity; optic atrophy; oedema of legs. *Case* 15: Headache, dizziness, weakness of legs, unsteady gait, blindness; spasticity; optic atrophy. *Case* 16: Abdominal pain, headache, paraesthesiae, weakness of legs, severe ataxia, impaired vision and later optic atrophy. *Case* 17: Colic, diarrhoea, polydipsia, paraesthesiae, general pains especially in legs; inability to walk; atrophy of muscles of legs, no fasciculation, spasticity of limbs. *Case* 18: Vomiting, diarrhoea, abdominal colic, inability to walk; then euphoria, delirium, and hallucinations. Irregular pulse. *Case* 19: Dizziness and fainting, then generalized tremors, spasticity. Psychotic. *Case* 20: Pains in hips, legs and shoulder; aching bones. *Case* 21: Intense pruritus then melaena, marked loss of weight. *Case* 22: Pain in hips, legs, then abdominal pain, paraesthesiae; cauteries in abdomen and knees for pain. Blue gums, septic teeth. *Case* 23: Pain in knees then generalized, oliguria, grey zone on incisors. *Case* 24: Epigastric pain, pain in shoulders, back and legs, severe in legs; vertigo; marked sweating. *Case* 25: Dark urine, general oedema, later ascites; pain in back, chest, and legs, cough. *Case* 26: Backache, then pain in limbs; after these subsided abdominal pain appeared; marked muscular twitchings; cough and expectoration.

presence of a trace of albumin in 58%, albumin + in 35%, and albumin ++ in 6% of the samples. Hyaline casts were seen in 22%, granular casts in 17%, and waxy casts in 5%. In 18% of the specimens a few red blood cells were seen. In severe cases the blood urea was much increased.

2. *Skin lesions.*—Pruritus is not uncommon, mainly in the palms, soles, and genitalia. In severe cases exfoliative dermatitis follows, especially of the hands and feet.

3. *Nervous lesions.*—These are almost constant in cases of all grades of severity. Loss of power in the legs and difficulty in walking or inability to walk are frequent. Cerebellar ataxia, disturbance of speech, paraplegia, or general spasticity with brisk reflexes are common. The plantars may be extensor while the abdominal reflexes are retained. Ankle clonus is occasionally present while tendon reflexes are lost. Restriction of visual fields or marked loss of vision with optic atrophy are not infrequent. Tremors or intention tremors are sometimes found. Para-

esthaesiae are noticed by some patients but sensation remains intact.

4. *Mental condition.*—Mentality is usually not affected, but some patients lost consciousness and then died. A few patients had headache, insomnia, confusion, hallucinations, or excitement, and one woman wanted to throw her child from the balcony. Manic states were also seen.

5. *Cardiac involvement.*—In severe cases the pulse is irregular, sometimes with bradycardia. Electrocardiographic examination of severe cases showed frequent ventricular ectopic beats, prolongation of Q-T interval, depression of the S-T segment, and T inversion.

6. *Deep skeletal pain.*—This is generally felt over the muscles or bones and is frequent, sometimes very severe and not alleviated by the usual analgesics. The pain is usually generalized but is sometimes confined to the lower extremities and less often to the shoulders. Fasciculation and coarse twitching of muscles are also present. Myoidema is elicited in many cases.

7. *Gastro-intestinal symptoms.*—These are usually not very severe. Vomiting and/or diarrhoea are sometimes present; some cases have constipation. Epigastric or hypogastric pain or colic are also frequent. Two cases had melaena, and another two had dysphagia.

The symptoms and signs of poisoning by ethyl mercury toluene sulphonanilide are different from those caused by methyl mercury described by Hunter *et al.* (1940). In their cases no abnormality was noted in the cardiovascular system, the visual fields being constricted, and the fundi remaining normal; while in our cases the heart was affected and optic atrophy seen. Also their cases did not experience the skeletal pain, muscular twitching, polyuria, or skin changes which were frequent in the present series. It is interesting to note that the members of some families all had similar symptoms; for example three brothers admitted to hospital all had optic atrophy.

Fatal cases had severe involvement of the nervous system or cardiac abnormalities evidenced by the irregular pulse or electrocardiographic changes. Mild and moderately severe cases recovered. Unfortunately, when patients left hospital they could not be traced and no adequate follow-up was possible. It is possible that the disease was more serious in the cases we report because the patients' general health had already been undermined by endemic diseases, particularly ankylostomiasis (Jalili and Demarchi, 1952), and the low protein, low vitamin diet on which they exist. Wheat and barley in the form of home-made bread provide a very large proportion of their diet (Jalili, Georges, and Fadhil, 1950; Jalili and Al-Kassab, 1959).

Some persons who consumed the dressed seed came to medical centres with no symptoms or signs; they sought medical advice because a member of their family had developed symptoms or had died.

An interval of a few weeks or months occurs between the distribution of the dressed seed and the appearance of cases of poisoning. This is partly due to the time needed for the cumulative effect of the poison to develop and partly to the fact that the farmers did not use the dressed seed for making bread immediately it was received, but only after their own stocks were exhausted, or after they found that others who consumed the seed had suffered no ill effects during the first few weeks.

Symptoms usually appeared within one or two months of eating the treated wheat, sometimes longer but occasionally in as short a time as a few days, depending on the amount consumed. Large amounts lead to the appearance of acute poisoning with gastro-intestinal symptoms and sometimes death.

The Electrocardiograms

Twelve-lead records were made for 15 patients; nine of these showed certain abnormalities (Table 2). The other six records were within normal limits. Impaired conduction evidenced by prolonged P-R interval, increased irritability of the heart with frequent ectopic beats, and severe myocardial damage shown by prolonged Q-T segment and S-T deviation with invertion of T waves were present singly or in combination (Figs. 1, 2, and 3).

Treatment

Supportive treatment with sedatives, analgesics, and vitamins was given. Pituitrin relieved polyuria and thirst in 10 out of 16 cases with improvement of the general condition (R. Tapponi, personal communication). ACTH and cortisone were not helpful.

Dimercaprol (BAL) is effective in mercuric bichloride poisoning and should be given early (Longcope and Luetscher, 1946; Troen *et al.*, 1951; Locket, 1957). Large doses are needed for the treatment of poisoning with mercury or its inorganic salts: 5 mg./kg. body weight as an initial dose followed by three doses of 2·5 mg./kg. body weight at three- or four-hourly intervals and a similar intramuscular dose given every six hours for the next 48 hours. Symptoms usually disappear within four weeks after contact with the poison. Neurological

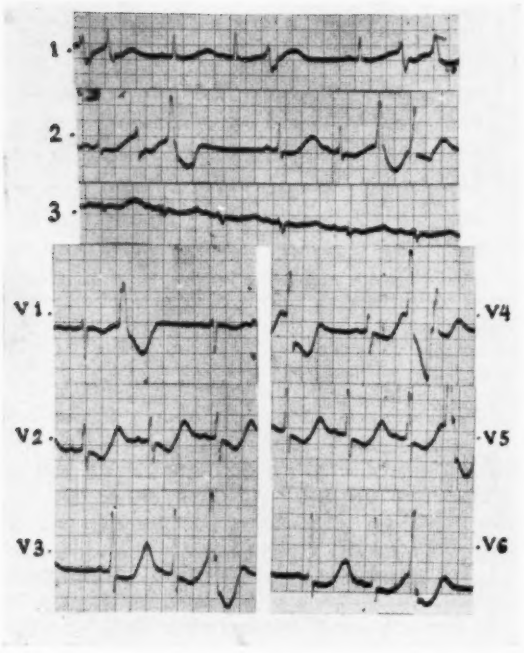


FIG. 1.—E.C.G. from a patient aged 35 years (Case No. 6). Symptoms of ethyl mercury toluene sulphonanilide poisoning of 10 days' duration.

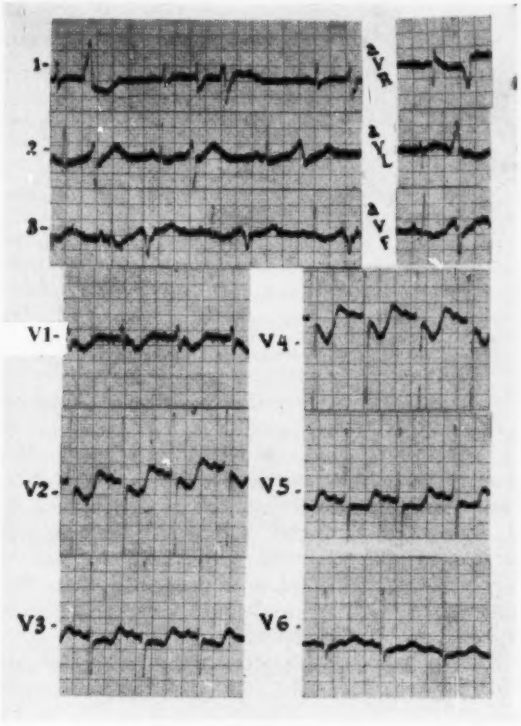


FIG. 2.—E.C.G. from a patient (Case No. 5) aged 14 years who had symptoms of poisoning for 14 days. She was seriously ill and died shortly after the record was taken.

Fig. 3.—E.C.G. from a patient 16 years of age (Case No. 17) with symptoms of poisoning of 15 days' duration who made an excellent recovery.

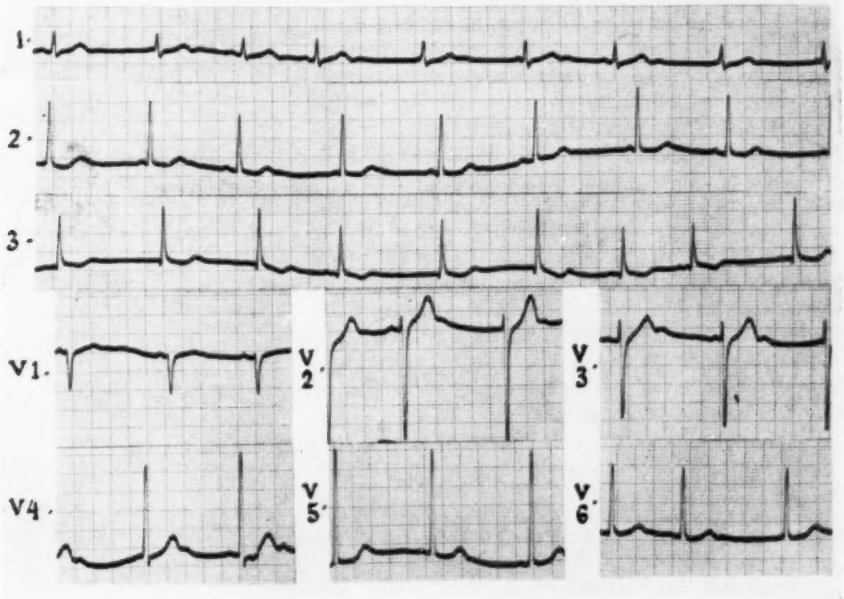


TABLE 2

FINDINGS IN ABNORMAL ELECTROCARDIOGRAMS

Case No.	Age (years)	Rate	Rhythm	Axis	Position	P-R (sec.)	Q.R.S. (sec.)	Q-T (sec.)	S-T	T
5 (Fig. 2)	14	72	Sinus*	Right	Vertical	0·16	0·09	0·28	Depressed in all praecordial leads	Inverted in VI, flat in I, II
6 (Fig. 1)	35	43	Sinus*	Left	Intermediate	0·18	0·08	0·60	Depressed in V2-V6	Flat in I, inverted in VI
8	10	140	Sinus	—	Intermediate	0·9	0·08	0·32	Normal	Inverted in III, VI, V2, V3, flat in V4
9	16	70	Sinus	—	Intermediate	0·14	0·08	0·52	Depressed in V4-V6	Inverted in all leads
10	23	100	Sinus	—	Intermediate	0·21	0·09	0·39	Normal	Normal
14	9	60	W.P.M.†	—	Intermediate	0·16-0·12	0·08	0·40	Normal	Normal, U wave in V2, V3
15	10	80	Sinus	—	Vertical	0·13	0·09	0·40	Depressed in I, II, III, V1-V5	Inverted in V1-V3
17 (Fig. 3)	16	60	W.P.M.†	Right	Vertical	W.P.M.	0·10	0·43	Normal	U-wave in all chest leads
18	35	120	Sinus*	—	Intermediate	0·16	0·09	0·48	Depressed in V1-V6	—

*Sinus rhythm with multifocal ventricular ectopic beats. Rate of dominating rhythm.
†Wandering pace-maker.

phenomena occasionally persist and may be due to continued unnoticed contact, psychological factors, or, rarely, to persistent damage of the central nervous system, as mercury has been detected in the cerebrospinal fluid a few months after the cessation of exposure. In chronic cases smaller doses of BAL may hasten recovery.

BAL is not expected to have any appreciable effect in poisoning with alkyl-mercury compounds because they produce toxicity as such and not as the result of the dissociated mercury.

BAL was used in many cases and our general impression is that it did not influence the course of the disease. However, it is difficult to ascertain its value where progress is so unpredictable. In five cases it had no appreciable effect, but in two cases it was accompanied by a rapid improvement in symptoms and signs. One of these had severe ataxia and was almost blind, but five days after the institution of BAL therapy he was able to walk well and his vision was greatly improved. BAL was given in a dosage of 100 mg. four-hourly for two days and eight-hourly for another 10 days. Apart from slight restriction of the visual fields and pallor of the temporal halves of the optic discs the patient made a complete recovery. In another case severe pain of sciatic distribution and exfoliative dermatitis of the legs quickly disappeared on BAL therapy. The pain did not respond to analgesics. Further experience with BAL in cases of poisoning by this type of mercury compound is needed.

Prevention

Organic mercury compounds are used as seed disinfectants of wheat, oats, other cereals, and for cotton and sugar beet. It is therefore necessary to take preventive measures against poisoning. The following measures are suggested:

1. Education of the population at risk using all available means.

2. The addition of a colouring material or a substance with an unpleasant taste to the fungicidal compound. This would make it almost impossible to use the dressed seed as food.

3. The protection of workers during dressing of the seed, which should be carried out in a completely enclosed apparatus.

4. Seed should be distributed immediately before the sowing season and used for this purpose only.

REFERENCES

Hunter, D., Bomford, R. R., and Russell, D. S. (1940). *Quart. J. Med.*, n.s., **9**, 193.
——, and Russell, D. S. (1954). *J. Neurol. Psychiat.*, **17**, 235.
Jalili, M. A., and Al-Kassab, S. (1959). *Lancet*, **2**, 108.
——, and Demarchi, M. (1952). *J. Fac. Med. Iraq*, **16**, 71.
——, Georges, F., and Fadhil, A. S. (1950). *Ibid*, **14**, 73.
Locket, S. (1957). *Clinical Toxicology*, p. 572. Henry Kimpton, London.
Longcope, W. T., and Luetscher, J. A. Jr. (1946). *J. clin. Invest.*, **25**, 557.
Lundgren, K-D, and Swensson, A. (1949). *J. industr. Hyg.*, **31**, 190.
Stoneman, M. E. R. (1958). *Lancet*, **1**, 938.
Troen, P., Kaufman, S. A., and Katz, K. H. (1951). *New Engl. J. Med.*, **244**, 459.
Williams, N. E., and Bridge, H. G. T. (1958). *Lancet*, **2**, 602.
Wilson, V. K., Thomson, M. L., and Holzel, A. (1952). *Brit. med. J.*, **1**, 358.

MISCELLANEA

The North of England Industrial Health Advisory Service

BY

R. C. BROWNE and D. GRIEVES

In January 1960, the North of England Industrial Health Advisory Service was launched. It is sponsored jointly by the trade unions, management, and King's College, Newcastle upon Tyne, with support from both the Nuffield Foundation and the Nuffield Provincial Hospitals Trust.

This new service offers industry consulting facilities which are not already covered by the National Health Service, at a charge of 2s. per employee per annum, with an additional charge, at cost price, for any special investigations carried out. Every item of work is carefully costed. Advice is not withheld from non-member firms, but it is given on a cost-plus basis.

The subscription is low, but it is calculated on a working population of about one million in a very heavily industrialized area.

The University has agreed that its Department of Industrial Health may loan its professor, senior lecturer, and lecturer in occupational hygiene to the service, for the time being. During the first year's work, moreover, a house has also been provided.

There is a Committee of Management of six members, composed of equal numbers of University, industrial, and T.U.C. representatives.

On the clinical side, the service advises about the resettlement of cases of long-term sickness, and, on the scientific side, it advises on the whole range of occupational hygiene and environmental problems. It possesses a fully equipped occupational hygiene laboratory under the direction of the University lecturer in occupational hygiene.

The growth of the service is expected to be slow, but during the first year's work, 16 firms of varying size, covering a wide variety of processes, became members. These firms have a total manpower of 7,500 workpeople, ranging from a very small firm with 24 employees, to one employing as many as 1,841. Although the very small firms have the greatest need of such a service, it has been found that they are the least likely to join. The most promising group seems to be that which employs between 100 and 500 workers.

A variety of interesting problems have already been dealt with for both member and non-member firms. These have included the investigation of a dust hazard in a cabinet-making factory, and a lead-in-air survey in a shipbreaking yard together with the urinary lead estimations of the men employed. A survey of the atmosphere of a department in an electrical engineering firm engaged in the manufacture of lead enamels, together with urinary lead estimations of the men employed, has also been made. The noise level has been estimated in the bottling department of a brewery, and a dust survey of the oil gasification plants in the North of England is in progress. A bulletin is issued by the service every six months.

The Committee of Management of this service have been eager to tailor it so as to provide, on a regional basis and at a price which firms in the North are prepared to pay, long-term sickness and occupational hygiene advice. Neither of these can be obtained from the National Health Service.

General Assembly of the Permanent Commission and International Association on Industrial Health

Two Sessions of the General Assembly were held in New York in July 1960, during the International Congress. The following information is extracted from the Minutes of these Sessions through the courtesy of the Secretary-General, Professor Enrico Vigliani. Sixty-four members attended each session. President Forssman opened the meeting and welcomed the members present. At the previous Congress in Helsinki, four sub-committees were appointed to consider the following subjects (the name of the chairman of each sub-committee is given in brackets).

Absenteeism (Professor Forssman, Sweden).—The Report of the Sub-committee will be published in the Proceedings of the Conference. This Report deals with definitions, recommendations, and information to be given when planning studies on absenteeism in industry.

Maximum Allowable Concentrations of Toxic Substances (Professor Truhaut, France).—An international symposium was held in Prague in 1959 under the sponsorship of the Permanent Commission and of the International Union of Pure and Applied Chemistry. Organized by Professor Teisinger, the particular aim of the symposium was to discuss the reasons for the profound differences in recommended maximum allowable concentrations between the various countries, with special reference to the U.S.A. and U.S.S.R. The resolutions of this symposium will be published by the International Union of Pure and Applied Chemistry.

Occupational Health in the Textile Industry (Professor Schilling, United Kingdom).—Twenty-two countries had been asked to supply data on the incidence of byssinosis in their national textile industry. From the replies received it was concluded that byssinosis occurs in at least eight countries and probably more. In 1959 a meeting on byssinosis was held in London, sponsored by the Medical Research Council with international attendance. In 1960 an international meeting was held in Milan on the pathogenesis of byssinosis and related conditions. Further meetings on the pathogenesis and prevention of the disease will be held during the next three years.

Constitution of the Commission (Professor Vigliani, Italy).—A revised draft of the constitution has been prepared. This was discussed and, as a number of matters remained outstanding, it was decided that the sub-committee should continue its work and prepare a final draft Constitution for submission to the next General Assembly. It was agreed that the title of the organization be changed from "Permanent Committee" to "Permanent Commission" and that the Bureau should be authorized to nominate new members of the Permanent Commission during the period between international congresses.

After discussion it was agreed that there should be no official journal of the Permanent Commission, and that information should be circulated to all journals in the field of occupational health.

It was decided to accept the invitation of the Spanish Government to hold the next Congress in Madrid in 1963.

Professor Sven Forssman was re-elected President for the triennium 1960-63. Professor R. E. Lane was elected a Vice-President on the nomination of the British Members. Also elected Vice-Presidents were Professor E. W. Baader (Germany), L. Greenburg (U.S.A.), A. A. Letavet (U.S.S.R.), L. Noro (Finland), and J. Teisinger (Czechoslovakia). Professor Enrico Vigliani was re-elected Secretary-General and elected Treasurer (Address: Clinica del Lavoro, Via S. Barnaba 8, Milan, Italy).

The British members of the Permanent Commission and International Association on Industrial Health are:—Professor R. E. Lane (Vice-President of the Commission); Dr. L. G. Norman (Chairman, British Members); Professor R. S. F. Schilling (Hon. Secretary, British Members); Dr. A. T. Doig; Dr. N. Langdon Lloyd; Mrs. I. G. Doherty; Dr. Donald C. Norris; Dr. Sibyl Horner; Dr. E. R. A. Merewether; Dr. J. M. Davidson; Dr. A. I. G. McLaughlin; Dr. M. W. Goldblatt; Dr. Donald Hunter; Dr. A. Lloyd Potter; T. E. A. Stowell, Esq.; Dr. A. Meiklejohn; Sir Walter Chiesman.

Two New Medical Centres

H. J. Heinz Co. Ltd.

A large new factory at Kitt Green, near Wigan, Lancs., was opened in 1959.

The following particulars were provided by Dr. J. C. Graham, Chief Medical Officer. The medical department provides a personal service for any employee who suffers from illness or accident at work; it is also concerned with environmental working conditions, efficiency of operation, and a high quality of product.

The medical department is situated in the Employee Services Building at ground level, opening on to the main personnel corridor next to the south entrance. It has a floor space of 3,360 square feet. A plan of the accommodation is given in Fig. 1.

The lay-out of the department was dictated to some extent by the site allocated to the medical services. Ideally, the medical department should be situated as near to the centre of a factory as possible to curtail the distances to be covered by employees requiring treatment; at the same time, it is necessary for it to be conveniently situated for access by new employees attending for medical examination. The decision, however, to locate all employee services in the one building, precluded any possibility of it being in the centre of the factory. Sited as it is, however, adjacent to other service departments, it has its own entrance and is a self-contained unit; this is important to preserve the confidential doctor–patient relationship.

To reduce noise interference to a minimum the examination suite faces an internal courtyard, whereas the surgeries and dental unit face south to give maximum daylight. The treatment rooms have tiled walls and floors; the other rooms are finished in plaster. All ceilings are covered with acoustic tiles. The artificial lighting is mainly by fluorescent tubes fitted with plastic diffusers giving an illumination of at least 30 lumens per square foot in the working areas. The lay-out is related to four separate functions of occupational health services.

Treatment and Casualty Service.—The rest rooms, surgeries, sterilizing and minor treatment room are designed for the treatment of all personnel with minor or major conditions arising at work. The larger of the two rest rooms has double doors opening directly on to the main corridor and surgery to allow easy access for a stretcher trolley. The minor treatment room is also intended for use as a recovery room linked with the work in the dentist's surgery.

The waiting area has been purposely kept small. This room also contains a refrigerator so that patients on a milk diet may receive their treatment without interfering with the general flow of patients.

The main surgery area (Fig. 2) is divided by a curtain

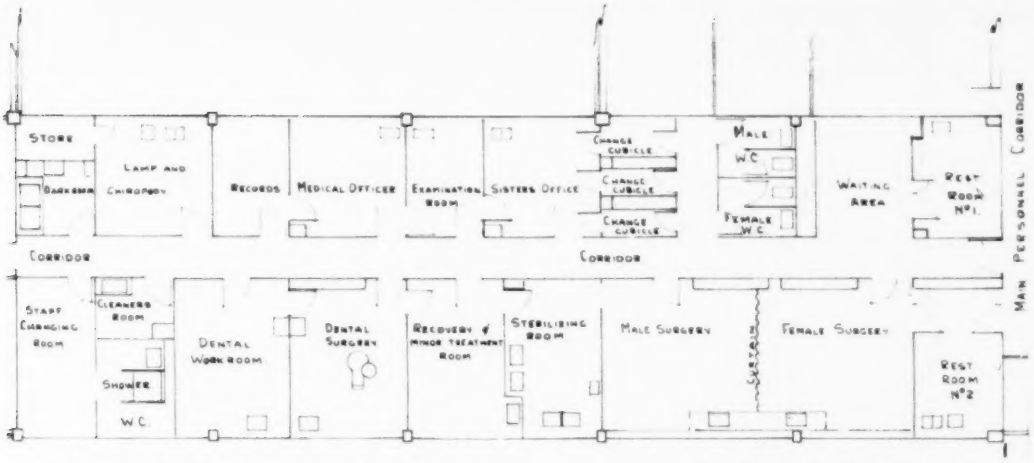


FIG. 1.—Kitt Green factory medical department. The department is approximately 100 feet in length and 34 feet in width.

separating the sexes, and a special area is set aside for the treatment of eye conditions; all eye treatments are given with the patient lying down.

Medical Examinations and Consultations.—The consulting suite is designed to allow a flow of personnel from the waiting area to the changing cubicles which, in turn, gives access to the sister's office where a trained sister can take the occupational and health histories and record the usual information before reference to the doctor who normally examines the patient in the examination room. The sister's office is also used for health counselling and for interviewing on return to work all personnel who have been absent due to sickness.

FIG. 2.—General view of surgery, Kitt Green. There is a separate sterilizing room.

Ancillary Services.—The dental unit consists of a surgery, workroom, and a dark room, since it is intended to equip the dental surgery with an *x*-ray machine, and, as mentioned above, the dentist has part use of the minor treatment room as and when required.

Across the corridor is a multi-purpose room for use by the chiropodist whose services are provided free to all employees. Minor physiotherapy treatment can also be given at the request of an employee's general practitioner.

Administration.—The records office is adjacent to and gives access to the medical officer's room and contains records of all employees; sickness absence statistics are compiled from these records. The nursing staff are provided with a changing room with shower accommodation.

Staff.—It is ultimately intended to provide services for 3,000 personnel; at present there are 2,200 employees and a 24-hour service is provided five days per week. The full-time medical officer and sister-in-charge are also responsible for the medical department at another factory of the Company seven miles away which employs a further 1,600 persons. Additionally, there are two state registered nurses and three state enrolled assistant nurses to provide the 24-hour cover. The medical officer has a full-time secretary who is responsible for the sickness absence records. A cleaner is also employed.

Richard Thomas and Baldwins Ltd.

A new medical centre was opened at the Ebbw Vale Works in February 1961. The following description was provided by Dr. J. S. Spickett, Chief Medical Officer.

A plan of the new centre is given at Fig. 3. The centre provides most of the services normally found in a hospital out-patients' department.

The central area of the building provides space for a waiting room and a large records office and placed around this are the consulting rooms and treatment departments. Three full-time doctors are based at the works and each has his own consulting room, with a curtained-off cubicle.

For minor injuries there is a large dressings room, and in this there are cubicles for minor surgical procedures, treatment of eye injuries and privacy for carrying out more elaborate dressings (Fig. 4).

Behind this is an ambulance drive-in and a resuscitation ward, with three beds, where more serious cases can be dealt with. Next to it is the *x*-ray department, fitted with a modern unit that ensures that the patient receives the smallest possible *x*-ray exposure.

In the records room preliminary particulars of newcomers to the Company are recorded by the nursing staff before examination by the doctor, and medical records of all employees are housed.

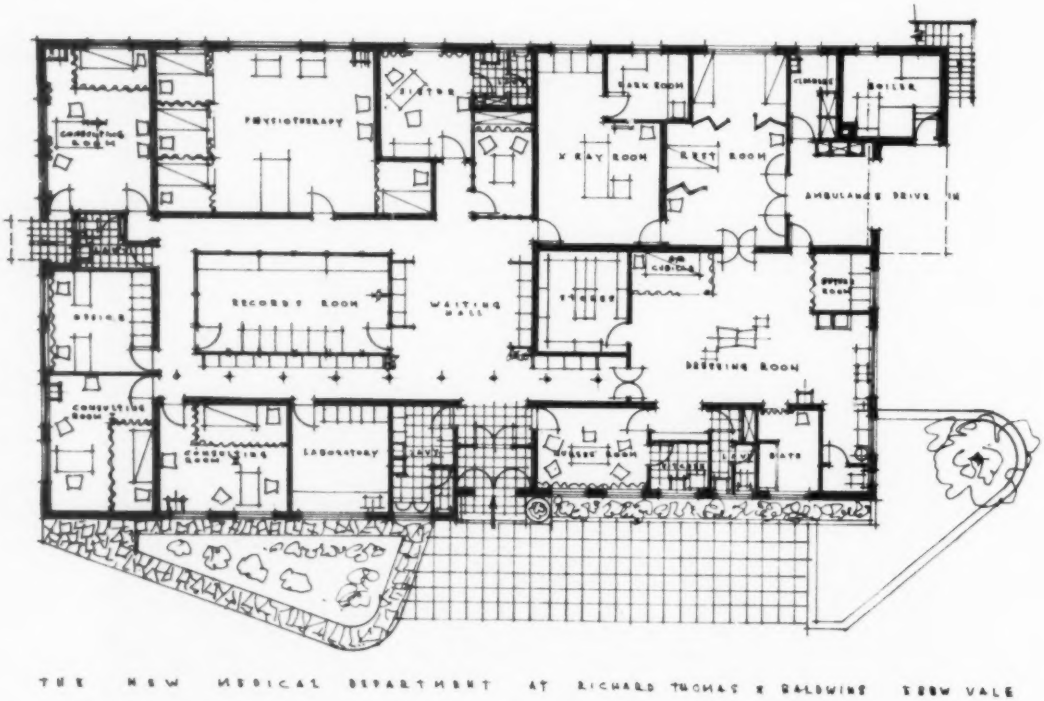


FIG. 3.—The new medical department at Richard Thomas and Baldwins, Ebbw Vale. The overall length of the front of the building is 102 feet, of the back 110 feet, and the width is 58 feet.

FIG. 4.—The main surgery has a "glasscrete" ceiling and walls of soft grey tiles. In the left background is the cubicle for minor surgical procedures.

A small sound-proof laboratory is provided for the use of the medical officers and as a base for the company's occupational hygiene unit, when engaged on investigations at Ebbw Vale.

The design artist responsible for the internal finishes has employed materials which can be maintained at a high standard of cleanliness. This has been achieved by using a limited number of paint colours, with wallpapers to give a texture effect, leaving curtains to introduce a note of colour where necessary. These are of a plastic material and are resistant to acid and chemicals. Black "granwood" flooring has been used except in the treatment rooms. The ceilings in the waiting area and treatment rooms are of acoustic tiles with recessed light fittings.

In the waiting hall area, one wall has been completely covered with plastic fabric with vertical aluminium cover strips to give a decorative effect emphasized by the use of glass pendant light fittings.

The built-in seating is covered with plastic material and provided with draught screens at each end in contrasting colours.

The dressings room (Fig. 4) has been tiled in soft grey, relief being given by the use of coloured curtains and contrasting coloured tiles in the adjoining spaces; the whole of this floor area is in dark terrazzo with brass inlay strips. Every window in the building is double glazed to reduce noise and dust to a minimum.

The staff of the medical department at Ebbw Vale consists of three doctors, a sister-in-charge, 22 nurses, eight rescue men, two hygiene supervisors and a physiotherapist. The three doctors also cover the Company's works at Panteg, Pontymister, and Abercarn, where there are permanent surgeries, all staffed by nurses.

The aim of the medical service is both preventive and therapeutic.

The preventive programme includes the selective placing of personnel, medical supervision aimed to maintain the highest possible standard of health, control of the environment, making use of an occupational hygiene team, and public health control in co-operation with local Medical Officers of Health.

The therapeutic programme, organized in co-operation with local general practitioners and hospitals, provides treatment for occupational injuries and illnesses and, in certain cases, for non-occupational conditions. Depending on local needs, special facilities such as resuscitation, radiology, physiotherapy, and rehabilitation are provided.

OBITUARY

Dr. J. A. A. Mekelburg died suddenly at London Airport on July 4, 1959. He had returned from a holiday in Northern Ireland after enjoying, with characteristic enthusiasm, the Belfast meeting of the Association of Industrial Medical Officers. He died as he would have wished, briefly and while still at work.

John (his full name was Johannes) Mekelburg was born in South Africa in 1899. He was educated at South African College School and Cape Town University. In 1919 he came to England and qualified in 1925 from Guy's Hospital. He settled in general practice at Forest Gate, London, where he worked until 1939, when he became medical officer to Messrs. Peek Frean and Company in Bermondsey. With this work he combined the duties of safety officer and developed an exemplary industrial medical service. His natural kindliness as doctor, friend, and wise counsellor brought help and relief to many employees and colleagues at difficult times.

John Mekelburg was a man of many interests. He was an international smallbore shot and won many prizes at Bisley, an enthusiastic collector of stamps, and a good amateur photographer. In 1957, with his wife and daughters, he was happily able to visit South Africa and enjoy the familiar places of his youth. The coloured slides he took on this journey were enjoyed by many friends. He was an active member of the Royal Society for the Prevention of Accidents and made a considerable contribution to the work of the Society.

John Mekelburg's heart was devoted to the Association of Industrial Medical Officers. He became Honorary Secretary of the Association in 1949 and was tireless in his personal efforts to secure its expansion and wider recognition. In 1949 the Association had 500 members and it was Mekelburg's ambition, which was realized in 1958, to increase this number to 1,000. He then gave up the post of Honorary Secretary to undertake a less arduous task as Business Manager of the Transactions. This was particularly appropriate for it was his enthusiasm that inspired the publication of the Quarterly Bulletin of the Association, the first number of which was published in September 1948. From this newsletter grew the Transactions. These are matters of history which will not be forgotten. But we shall not forget also the high integrity and dedication of John Mekelburg to industrial medicine. To his friends he was kindly, sincere, and loyal, a colleague whom we shall sadly miss. John's life was well and fully lived; his death will be felt as a personal loss by all his colleagues. Our sympathy goes to his widow and two daughters, who often accompanied him to the Association meetings he enjoyed so much.

L. G. NORMAN

BOOK REVIEWS

Classification of Occupations 1960, General Register Office. (Pp. 136. 27*s*. 6*d*.) London: H.M.S.O.

This volume is compiled primarily for use in the Census, but it is also the principal mode of categorization in the Registrar General's Occupational Mortality Supplement, in morbidity inquiries, and in various small-scale and local studies, hence its value to research workers and industrial medical officers. The social class groupings are used in much socio-medical research thus ensuring a degree of comparability between studies rarely achieved in the field of social differentiation. For these and other reasons there will be considerable interest in the new classification.

The reduction in the number of occupational groups constitutes the largest change in the system of classification since 1921; this in itself raises a number of considerations only a few of which can be touched upon here. The allocation of occupations to occupational groups, and of occupational groups to social classes has been brought up to date but certain anomalies remain. The job descriptions "sandblaster"—which exists no longer—and "drowner" are not to be found in the alphabetical index; the civil servant has been given a code number to himself while the shepherd and the greengrocer have been robbed of theirs; the van driver turned instructor does not lose status and the doctor who passes from university employment to industry is no longer promoted.

The experts came to the conclusion that "the degree of sub-division in 1951 and earlier classifications was over-elaborate, having regard to the quality of the answers given at the Census or death registration, for the main purpose of the Census and occupational mortality analyses". Few of us are qualified to dispute the validity of this statement where Census and mortality data are concerned, but from the practical point of view there are a number of drawbacks to the present system: there is an increased likelihood of occupational hazards being hidden and comparisons over time will be even more difficult to interpret than before. The increased heterogeneity of many of the occupational groups—there are now about 200 instead of about 600—will make further grading of occupations as by nature of work (Morris and Crawford, 1958) more difficult. The increased insensitivity of the classification may encourage those working on special inquiries to rely to a greater extent on groupings of their own construction.

The orders under which the occupation groups are enumerated have remained substantially the same, but the sub-orders have been dispensed with. The detailed "Classified List" has been replaced by a 12-page description of the contents of each of the occupational groups. Since the coder can pass, as previously, direct from the alphabetical index of occupational terms to the occupation groups and class categories this need not concern him greatly. For the social class allocation he must now turn to the Appendix; here it is combined with employment status and socio-economic group. The layout of the alphabetical index is little altered, but the introduction of italics for the industry, where entered, makes for greater clarity. This volume is more manageable than its immediate predecessor, but some valuable detail has been lost.

Since considerable changes have been made it is perhaps regrettable that they are based primarily on one set of considerations, *i.e.* the need to reduce registration anomalies, and that no allowance has been made for new developments and changing emphasis in epidemiological and social inquiry.

REFERENCE

Morris, J. N., and Crawford, M. D. (1958). *Brit. med. J.* 2. 1485.

SUSAN MEADOWS

Klinische Grundlagen der Sechsundvierzig Meldepflichtigen Berufskrankheiten (The Clinical Bases of 46 Prescribed Occupational Diseases in West Germany). By Ernst W. Baader. (Pp. xii + 479; 90 figures. D.M.58.) Munich and Berlin: Urban and Schwarzenberg. 1960.

To those who remember Baader's little book of 30 years ago, the present volume of nearly 500 pages will inevitably bring a certain nostalgia for the days when one could carry extant knowledge of the subject with some measure of assurance and authority.

Baader himself, by his presence at conferences both here and on the continent, brings back more than a taste of the pioneers of those early days of the century. His manifest enthusiasm and brightly acute interest in all that pertains to occupational medicine is clearly recognizable in this greatly enlarged edition of the original work.

Now that German is much more widely understood and read in medical circles, there is no good reason for our colleagues in industry to miss the great experience of reading Baader's account of his vast experience in our field.

Before dealing with the 46 prescribed occupational diseases, Baader warns us that "Those who regard biological science as a mathematical affair will, in occupational medicine, soon founder". This is perhaps a useful reminder that Medicine, in all its branches, is an Art, albeit an Art which is always striving to become a Science. But the proposition as stated by Baader will not do, since it will tend to absolve practitioners of our subject from the onus of developing a reasonable competence in elementary statistics. The day is past when the legal

prescription of an occupational disease can be based upon isolated clinical observations only. We live now in an era of planned observation of populations. It is difficult enough to establish a non-specific disease as an occupational disease in an industry by carefully planned statistical record, without wasting time and effort on the collection of clinical cases devoid of the necessary data from which valid conclusions can be drawn. It is not necessary to designate such work as mathematical.

Baader rightly deplores the failure of physicians and surgeons to inquire into the occupation of their patients and instances cases where mis-diagnoses led to a continuance of hazardous work.

He rightly condemns the unrewarding diagnosis of gas-poisoning and the ambiguous designations of occupations, which do not reveal the nature of the patient's occupational exposure.

He approves the statement of Flury that poisons only "stimulate or inhibit the function of single organs and do not produce functions which are normally not characteristic of them" and illustrates from the chronic nephritis of lead, syphilis, vascular disease, or the end-stage of acute nephritis; from bronchitis and bronchopneumonia brought about by irritant gases or infection; from bladder tumours of aniline workers (*sic*) and bronchogenic cancer among miners of pitch-blend; from disturbances of heat regulation by CO, CS_2, CCl_4, and C_6H_6 which might be attributed wrongly to infection.

It must be admitted that the doctrine is at present almost unassailable but must ultimately be annihilated as more and more knowledge is gathered of the intracellular mechanisms of toxic action. The crude methods of today must soon be replaced by ways of distinguishing between, for example, the cellular disturbances brought about by different carcinogenic agents even though the ultimate tumours induced are clinically and histologically indistinguishable. It is true that there are more ways than one of killing a cat but the ways are distinguishable and more or less avoidable. The differences in reversibility of the effects of toxic agents should give us pause in equating the intermediate stages even when the final stages may appear identical. The long latent periods of many occupational diseases, the apparently contradictory results of the same toxic agent, the loss of resistances to many chemical noxa after more or less lengthy exposure, all await explanation which Flury's doctrine does not help us to attain. But he was writing over 30 years ago.

Baader gives the 1952 lists and the projected lists of prescribed diseases enacted in the German Federal Republic; the 1957 list in the East German Republic; lists in the Saar (1954), Austria (1955), and Switzerland (1952, 1956). The German lists are generally similar to those in the United Kingdom. The prescription of tear-fractures of spinal processes and of asbestosis combined with lung carcinoma rings strangely in British ears.

A difference from the more specific prescription used in this country for tumours of the bladder is the German formula of "Cancer or other new growths as well as changes in the mucous membrane of the urinary tract brought about by aromatic amines".

Another aspect of some interest in German prescriptions is the inclusion of the phrase ". . . which necessitate a change of occupation or complete cessation of all work". It may not be generally known that prescription in Switzerland includes a list of organic and inorganic recognized causes of "dangerous illnesses", and block designations such as "Chlorinated ethers of the aliphatic series" or "Halogenated aliphatic hydrocarbons".

The treatment by Baader of the extensive field from the clinical point of view is, as would be expected, comprehensive and in the classical manner.

The book is not easy to read consecutively but of its value as an up-to-date reference volume there can be no doubt.

The literature quoted is extensive even if, except in one or two fields, Britain does not figure very noticeably.

Professor Baader is to be congratulated on a notable achievement.

M. W. GOLDBLATT

Occupational Disease in California Attributed to Pesticides and Agricultural Chemicals, 1959. (Pp. 30.) California: State Department of Public Health. 1961.

Under Californian law a doctor who attends a patient injured at work and whose disability extends beyond the day of the injury must file a report. A total of 1,093 such "first reports" on cases of illness attributed to pesticides during 1959 are analysed and discussed in this publication.

About 42% of the cases were poisoning by organophosphorus insecticides. The only fatalities during the year were two men engaged in fumigation, one with carbon tetrachloride and the other with methyl bromide. Although "first reports" may not always give the final diagnosis these data from one of the most intensively developed and regulated agricultural areas of the world are instructive.

Complimentary copies are available upon request, within limitations of the supply, from the Bureau of Health Education, California State Department of Public Health, 2151 Berkeley Way, Berkeley 4, California.

J. M. BARNES

Micro-organisms as Allies: The Industrial Use of Fungi and Bacteria. By C. L. Duddington. (Pp. 256; 21 figures. 25*s*.) London: Faber and Faber. 1961.

This book deals with the many and varied ways in which bacteria and moulds are used in industry. The opening chapter entitled "The Livestock of Industry" describes the work of Louis Pasteur, generally accepted as the pioneer in this field, and details the research he carried out for the French wine and silk trade and how vast industries have been founded on the efforts of Pasteur and subsequent microbiologists.

As might be expected, a considerable amount of space has been given to the brewing process, and the yeast used in this trade is cultured with great care in very elaborate laboratories. The main differences between top fermentation, where the yeast grows on the surface of the wort to produce most types of British beers, and bottom fermentation, where the yeast lives at the bottom

of the vat under anaerobic conditions, yielding lager beer, are explained.

It is interesting to read that the wine trade has such antiquity dating from Mesolithic time, more than ten thousand years ago, and to follow its progress to modern commercial production where countries and districts specialize to market a characteristic product.

The diseases which affect the vine are many and varied and, until the advent of the newer insecticides and fungicides, the struggle has been intense, with the vine grower barely holding his own in this war of attrition. The commercial fermentation of the juice of the grape to yield wine is performed by the wine yeast *Saccharomyces ellipsoideus* and the growth of the wild yeast and bacteria is discouraged by the addition of sulphur dioxide to the "must" (grape juice) before fermentation.

The section of the book which deals with the famous vintage wines is fascinating and differences in vines, soils, and climates, which combine to give the characteristic types of great wines, are stressed.

It is recorded that Arrach, a spirit made by distilling the juice of the coconut palm, was known in India at least 800 years before the Christian era.

The Irish are given the credit for being the first race to distil whiskey from fermented grain and this preceded the production of brandy by the French. The legend has it that the Irish were originally taught whiskey-making by St. Patrick.

The use of yeast by the baker to lighten his bread has considerable antiquity, as the process was developed by the ancient Egyptians and the same method is used commercially today in all countries of the world.

The chapter on moulds and antibiotics chronicles the tremendous developments in this field in recent years, since the original observation of Fleming when he described the inhibition of cultures of *Staphylococcus aureus* by Penicillium. However, it is pointed out that the Chinese used soy flour poultices to treat infected wounds and the Ebers Papyrus of 1500 B.C. extolled the virtues of "yeast of sweet beer" for similar use.

This book is of importance to doctors who work in industry for great care has been taken to give a concise and accurate description of all the processes in which micro-organisms play a part in industry.

No mention has been made of any occupational diseases attributable to these allies of industry but no doubt the author would consider such information as outside his terms of reference.

JOHN F. EUSTACE

Toxicology and Biochemistry of Aromatic Hydrocarbons. By H. W. Gerarde. (Pp. xiii + 329; 89 figures + 63 tables. 30*s*.) Amsterdam: Elsevier. 1960.

This book is divided into two parts. The first, General Considerations, includes a brief account of the nomenclature and structure of the aromatic hydrocarbons, also the relationship between chemical constitution and physical properties, taste, smell, toxicity, and biochemistry. The biochemistry of these compounds includes sections on absorption, tissue distribution, excretion, and metabolism. The final chapter of the section discusses what constitutes an industrial hazard, threshold limits, and treatment.

The second part deals in more detail with 20 aromatic hydrocarbons commonly encountered industrially, under the headings "physical and chemical properties", "sources and probable modes of contact", "analytical methods", "toxicity", "biochemistry", "prevention, detection and treatment of exposure". A brief section on the carcinogenicity of the polycyclic hydrocarbons is included, and an appendix containing a glossary of terms commonly encountered in the petroleum industry.

The industrial hygienist and toxicologist, for whom it is primarily intended, should find this a valuable handbook, containing in one volume much of the chemical, biochemical, and toxicological data he requires, set out in tabular form and including previously unpublished work of the author.

The book is printed on good quality paper, but the binding of the review copy was inadequate, loose pages appearing after minimal usage.

C. J. THRELFALL

Air Pollution. (Monograph Series No. 46.) (Pp. 442; 4 figures. 40*s*.) Geneva: World Health Organization. 1961.

In the historical review by E. C. Halliday which opens this Monograph there is a clear indication of the rapidly mounting number of papers on air pollution which have been published since the end of the Second World War. There have as yet been few books summarizing these developments, but this well-produced volume goes a considerable way towards bridging the gap. It is intended mainly for the benefit of public health officials and it brings together a series of 14 articles by authors from seven different countries. It suffers from the inevitable time lag involved in the preparation of books with multiple authorship, but it provides a useful survey of world trends in research up to 1957. The coverage is wide, but for British readers it is liable to set a somewhat false perspective. Care has been taken to exclude purely occupational hazards, but throughout there is a tendency to emphasize the importance of industrial emissions. Domestic sources of pollution receive very little attention and nowhere is there any detailed account of the distribution of smoke and sulphur dioxide within large towns.

The individual sections vary widely in length and quality. Some contain a profusion of detail worthy of a reference work whilst others are written in terms too general even for a broad survey. One of the longest and most valuable is by Morris Katz, concerning the physical and chemical nature of air pollution. This is an admirable discussion of the several types of pollution which may be found in urban atmospheres with particular reference to conditions in Great Britain, Los Angeles, and the Detroit—Windsor area. It is followed by an equally long discussion by Harry Heimann on health aspects in which considerable emphasis is placed on the major episodes of high pollution. The amount of overlap which occurs between sections is quite small, but a table showing the average composition of the atmosphere appears in each of these two contributions and the Los

Angeles smog problem is discussed in several places. A section on meteorology by Harry Wexler sets out in quite simple terms the principles underlying the dispersion of pollution. Translation difficulties may account for a few errors which appear in the section on sampling and instrumentation by F. Cambi (*e.g.* the term "colloidal matter" may mean collodion). Not all of the recommendations in this section are appropriate for the study of general air pollution. Many of the methods described are intended for industrial dusts and the sampling and analysis of urban smoke is not properly considered. There is a brief but useful section on the effects on animals by E. J. Catcott and a very well-documented account of the effects on plants by Moyer D. Thomas. This describes the effects of sulphur dioxide, fluorides, and "Los Angeles smog" on sensitive plants and it includes some excellent coloured plates. Legislation is dealt with by Albert Parker, and although he refers to the several Acts which have been passed in Great Britain, the implications of the Clean Air Act, 1956, seem sufficient to justify longer discussion. There is an excellent survey on radioactive pollution by J. P. Jammet, although it goes far beyond the field of practical interest at the present time. It is wise to consider possible hazards from a wide range of radionuclides, but little information is given regarding their actual occurrence in the air and nowhere is the naturally occurring activity of radon daughter products used as a yardstick.

Other sections deal with the identification of the air pollution problem (Louis C. McCabe), economic and social aspects (E. Leclerc), site selection (J. R. Taylor, A. Hasegawa, and Leslie A. Chambers), prevention and control by process changes (Andrew H. Rose Jr., David G. Stephan, and Robert L. Stenburg), and fuel selection and utilization (K. Barker and W. A. Macfarlane). Each section has its own bibliography, extending to over 400 references in one case. There is an index which successfully links similar topics in different sections and, taken as a whole, this inexpensive volume can be recommended as a useful introduction to the subject to those with more than a passing interest in air pollution.

R. E. WALLER

Efficiency and Effort—An Analysis of Industrial Administration. By W. Baldamus. (Pp. viii + 139. 18*s*.) London: Tavistock Publications. 1961.

The thesis of this book is that industrial organization is inherently unjust since it attempts to impose a disparity between the amount of effort put out by the worker and the wages he receives; industrial unrest on this assumption, so far from being the exception, must be the most characteristic feature of industry. In order to discover the nature of effort, work is analysed into *work realities*, which are the physical working conditions, repetitiveness, and coercive routines, *deprivations*, which are impairment (*i.e.* actual physical discomfort caused by work), tedium, and weariness, and finally there are what Mr. Baldamus reluctantly describes as *relative satisfactions* including inurement, traction, and contentment —the latter term denoting the absence of unpleasant feelings rather than the presence of good ones.

As wages are costs to the firm, so the deprivations inherent in effort represent "costs" to the employee for which he deserves remuneration whilst being continually bilked by the employer who attempts to lower the value of effort. In the process of putting forward these views the writer demolishes, or attempts to demolish, almost every concept in industrial psychology since Elton Mayo: that work can be really satisfying, that high employee morale results in greater efficiency, that formal and informal structures in the firm are of any significance, and so on. As for improving human relations and social satisfactions, their main function is ". . . . to conceal from the worker precisely those managerial objectives which amount to increased wage disparity".

J. A. C. BROWN

Absence from Work: Incidence, Cost and Control. (Pp. 53. 10*s*. 6*d*.) London: British Institute of Management. 1961.

This booklet was prepared under the guidance of a committee consisting of representatives of the Institute of Cost and Works Accountants, the Institute of Personnel Management, and the British Institute of Management. It is anonymous, however, so that it is not possible to examine the statistical or medical qualifications of the authors.

The section on Incidence deals with the experience of manual workers in some 70 companies in the 12 months ended June 1956 and with the effect on absence rates of sex, seasonal influences, length of working week, size of firm, industry group, and locality. The interaction of the last four factors suggests that the conclusions based on this small sample of firms should be viewed with caution. The figures are not standardized for age and the effect of age on absence is not studied in detail.

The section on Costs relates to studies in 11 companies. The results varied widely; the total cost of absence to these firms ranged from 9d. to 15s. per week for each employee.

The section dealing with methods of controlling absence in five organizations is based on interviews with Officers of the firms concerned, and is almost entirely descriptive.

There is a useful bibliography.

C. J. CORNWALL

The Health of Executives. (Transactions of the Second Conference November 1960.) (Pp. 112. 15*s*.) London: The Chest and Heart Association. 1961.

This is an account of the proceedings of the second conference in the Royal Festival Hall promoted by the Chest and Heart Association.

For the industrial physician it contains very little with which he should not be already familiar. For the executives concerned, it must have been a stressful day, with 11 para-medical discourses to absorb, followed by question time. It might be asked for what purpose the Chest and Heart Association (formerly the National Association for the Prevention of Tuberculosis) gathers executives into the arms of its wisdom, when so many

diseases of managers arise roughly above or below C 7 and D 10!—and mostly above C 1.

The papers themselves make good reading on the whole and are not intended as significant contributions to the literature concerning degenerative and stress disorders, which are sometimes said to haunt our managers; those by Dudley White, O'Neil, and Norman probably best serve their purpose.

Sinclair (page 25) makes the somewhat controversial statement that "it is more dangerous to be ten pounds or more over-weight than it is to smoke heavily". This may be so, or may not, but it would be useful to have clarification as to what he means by smoking heavily and what is to be regarded as "over-weight"; standard tables relating height, age, and body weight are hopelessly misleading.

Finally, the uncharitable thought recurs constantly that if our managers were more scientifically selected and trained from youth, they would not need mid-career health education of this sort. Until that happy time this booklet will have to suffice.

OWEN MCGIRR

Rehabilitation of Hand Function. By A. N. Leont'ev and A. V. Zaporozhets. (Pp. 199. 60*s*.) Oxford: Pergamon Press. 1960.

The theme of this work is indicated by the first sentence of the foreword (by Colonel General of the Medical Service, E. Smirnov): "The main task of Soviet medicine at the present time is the eradication of the consequences of war." Many cases are described of men who were wounded in 1942-44. Presumably they had their rehabilitation long ago and it is difficult to understand why this work should be published in English so long afterwards. The latest reference is dated 1945. A publisher's notice apologizes for the poor quality of production "in the interest of speedily making available the information contained". There may be useful information in this work but it is difficult to find on account of the turgid style and poor typescript. No good service has been rendered to Soviet medicine by the publication of this work.

L. G. NORMAN

Injuries and Infections of the Hand. By R. H. C. Robins. (Pp. 220; 157 figures. 63*s*.) London: Edward Arnold. 1961.

This book adds another page to the better understanding of the hand and the modern development of treatment and rehabilitation. A brief reference to surgical anatomy precedes a review of general principles and due emphasis is laid on sound primary wound toilet. Prophylactic antibiotics are advisedly not recommended in all cases.

The importance of an early accurate assessment is stressed and the repair should be related to the patient's need to return to his pre-accident work.

Operative technique emphasizes the delicate handling of tissues and the use of fine instruments necessary to reduce the incidence of operative trauma and the limiting factor of fibrosis.

The chapter on infections deals with the more common conditions and their treatment. The author favours general anaesthesia, although local anaesthesia can be safely used for distal infections. Bed rest is ideal, but unfortunately accommodation in hospital is not always available for all hand infections. Tuberculosis of the hand is fully discussed. Accepted methods in the treatment of fractures and dislocations are well described.

The primary treatment of open injuries and skin cover by grafting is given well-deserved emphasis and the author has done a service in presenting so clearly the need to regard good skin cover as top priority in treatment.

This book is well illustrated and easily read, and reviews, in relatively few pages, the modern treatment of the hand, particularly in regard to injury.

It can be read with benefit by surgeons and house surgeons.

STEWART HARRISON

The Practical Management of Head Injuries. By John M. Potter. (Pp. xii + 84. 12*s*.) London: Lloyd-Luke. 1961.

This small book is one of those rare gems of common sense which cannot fail to absorb the interest of those who read it. It will prove of particular benefit to senior medical students, senior nurses, house surgeons, and especially to those often overworked young men who for the most part run our casualty departments. But no doctor who has to do with head injuries at any stage will regret the hour or two spent in reading through this little work.

The book is set out in orderly sequence of management: the casualty department, the ward, complications, convalescence, and rehabilitation, ending with three short appendices. At each stage the observations necessary and the practical steps to be taken are stated, and the reasons why they are important made quite clear. Esoteric descriptions of pathology and neurology in head injuries have been avoided, but the warning signs of impending serious complication are lucidly explained and the action required indicated with clarity.

Of especial interest to readers of this journal is the short final chapter on convalescence, rehabilitation, and sequelae. Here all the salient problems of after care in relation to occupation are discussed with masterly economy of words.

There is very little to criticize. The *early* establishment of a clear airway in the unconscious patient could perhaps be better emphasized. Some reference to brain stem compression at the tentorium, and to the reasons why lumbar puncture can be dangerous would perhaps have been useful elaborations.

Mr. Potter's style is everywhere most readable and concise. Among his acknowledgements he notes his affection for the work of Percival Pott, and running through the pages of this book there is discernible the same practical good sense that characterizes the writings of that great surgeon.

B. J. HARRIES

X-rays—Their Origin, Dosage and Practical Application. 8th ed. By W. E. Schall. (Pp. xi + 348; 356 figures. 50*s*.) Bristol: John Wright. 1961.

The 8th edition of this well-established book will be welcomed by those radiographers beginning a career in medicine or industry.

The material is set out in a logical fashion with clear diagrams and excellent photographic plates of a wide range of apparatus in common usage The text is, on the whole, readable and lucidly expressed, although in isolated instances, apt to be confusing.

The book covers the basic principles of radiation physics adequately although the sections dealing with supervoltage apparatus could well be amplified in view of the increasingly more widespread use and importance of this type of equipment. The retention of unit skin dose is hardly justifiable in this era of megavoltage therapy with its skin sparing effect.

The section on page 278 dealing with dose estimation from multiple fields should be amplified and revised and it is difficult to follow the argument advanced for the use of the "Auxiliary Concentrating Filter".

These are not major defects in what is certainly a valuable book of its kind.

The publishers have maintained a high standard of production and the book will deservedly retain its popularity amongst those for whom it was written.

B. STRICKLAND

BOOKS RECEIVED

(Review in a later issue is not precluded by notice here of books recently received.)

The Medical Management of Acute Poisoning. By Gordon Cumming. (Pp. ix + 128; with tables. 10*s*. 6*d*.) London: Cassell. 1961.

The Air We Breathe. A Study of Man and His Environment. Edited by Seymour M. Farber and Roger H. L. Wilson, in collaboration with John R. Goldsmith and Nello Pace. (Pp. xv +414; illustrated. 112*s*.) Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 1961.

Cell Function. By L. L. Langley. (Pp. xii + 377; illustrated. 60*s*.) New York: Reinhold; London: Chapman and Hall. 1961.

The British Steel Castings Research Association, Eighth Annual Report, 1961. (Pp. 37; illustrated.) Sheffield.

Good Health in the Tropics: Advice to Travellers and Settlers. By W. H. Jopling. (Pp. 31. 3*s*. 6*d*.) Bristol: John Wright. 1961.

Occupational Health Nursing. By F. H. Tyrer. (Pp. x + 173; 19 figures. 25*s*.) London: Baillière, Tindall and Cox. 1961.

Export Assistance Register. Foreword by C. O. Stanley. (Pp. 59.) London: Institute of Directors. 1961.

Report on the Health of the Royal Air Force and Women's Services of the Royal Air Force for the Year 1959. (Pp. 60; 19 tables + 5 charts.) London: Air Ministry. 1961.

The Health of People Who Work. Edited by Albert Q. Maisel. (Pp. xiii + 268. 40*s*.) New York: The National Health Council; Oxford: Pergamon Press. 1960.

The Nuffield Foundation, Sixteenth Report. Report for the Year Ended 31 March 1961. (Pp. xv + 210; 1 plate.) London.

Health in Industry. A Guide for Engineers, Executives and Doctors. By R. C. Browne. (Pp. vii + 157; 61 figures + 8 tables. 18*s*.) London: Edward Arnold. 1961.

The Relation Between Pneumoconiosis and Environmental Conditions. An Analysis of the Results of the First Series of X-ray Surveys in the National Coal Board's Pneumoconiosis Field Research. By D. Hicks, J. W. J. Fay, J. R. Ashford, and S. Rae. (Pp. viii + 42; 23 figures + 31 tables + 14 appendices.) London: National Coal Board. 1961.

Committee on Chronic Rheumatic Diseases. Report April, 1961. (Pp. 5.) Royal College of Physicians of London. 1961.

Symposium on Dust Control in the Pottery Industry, April 8, 1960 (Spec. Publ. 27). (Pp. 95; illustrated.) Stoke-on-Trent: The British Ceramic Research Association. 1961.

Basic Nursing Education Programmes. A Guide to their Planning. By Katharine Lyman. (Wld Hlth Org. Public Health Papers No. 7.) (Pp. 81. 5*s*.) Geneva: World Health Organization; London: H.M.S.O. 1961.

Teaching of Psychiatry and Mental Health. (Wld Hlth Org. Public Health Papers No. 9.) (Pp. 186; 2 figures + 9 tables. 10*s*.) Geneva: World Health Organization; London: H.M.S.O. 1961.

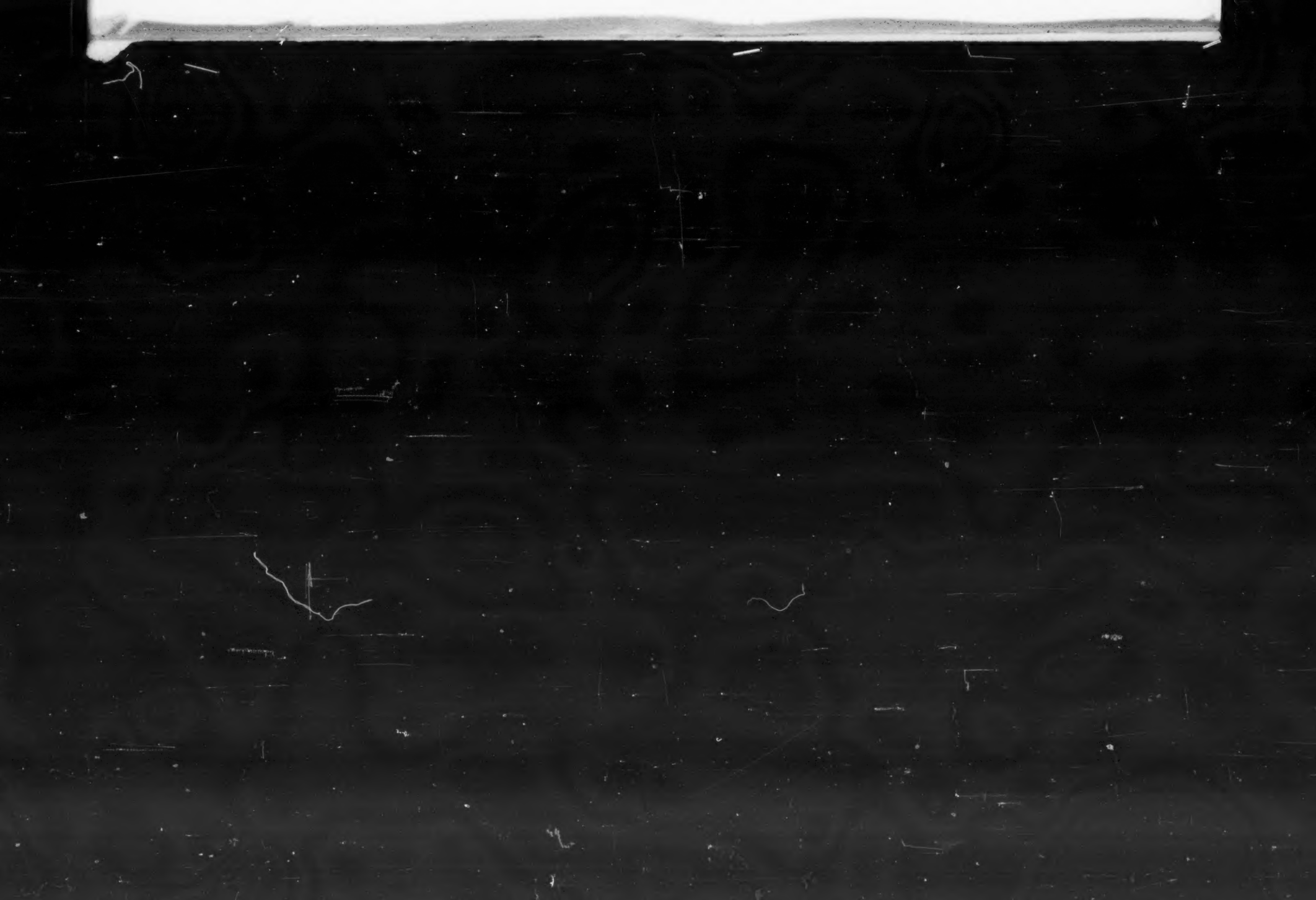
Proceedings Thirteenth International Congress on Occupational Health, July 25-29, 1960. (Pp. xxx + 1,005; illustrated.) New Jersey, U.S.A. 1961.

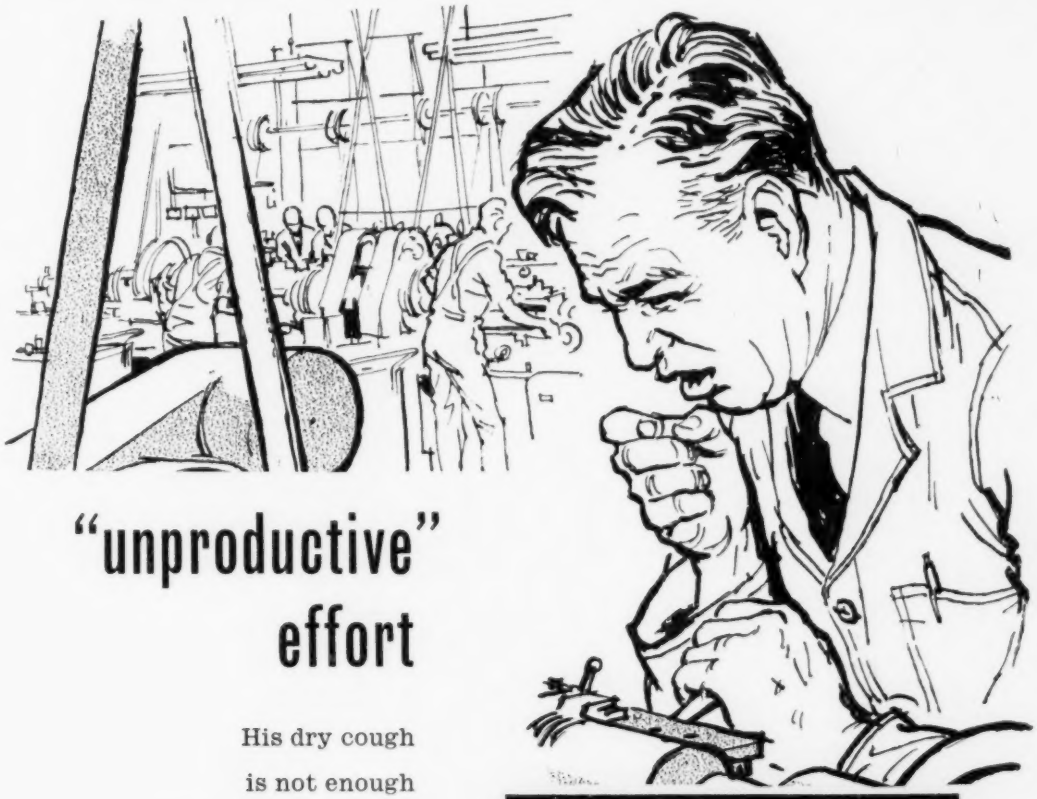
Principles of Medical Statistics, 7th ed. By A. Bradford Hill. (Pp. ix + 367; illustrated. 12*s*. 6*d*.) London: The Lancet. 1961.

Inhaled Particles and Vapours. Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Oxford, March 29 to April 1, 1960. Edited by C. N. Davies. (Pp. xi + 495; illustrated. 100*s*.) Oxford: Pergamon Press. 1961.

Report on the Health of the Army 1957. Part I (Statistical Tables). (Pp. iv + 78.) London: The War Office. 1961.

INDEX
PAGES
REMOVED





"unproductive" effort

His dry cough is not enough to keep him from work, but it wastes time and lowers his efficiency.

'PHENSEDYL'
trade mark *brand*

PROMETHAZINE COUGH LINCTUS
(with promethazine and codeine)

'VALLEDRINE'
trade mark *brand*

COUGH LINCTUS
(with trimeprazine and pholcodine)

For the relief of various types of cough, including the refractory post-influenzal type and bronchitis.

MANUFACTURED BY
MAY & BAKER LTD

Distributors: PHARMACEUTICAL SPECIALITIES (MAY & BAKER LTD) · DAGENHAM
M&B *brand Medical Products*

MA8341

VELROC*
plaster of paris bandages

Top of the bill with a strong supporting cast—VELROC Plaster of Paris Bandages. A strong, quicker setting, creamy plaster, firmly bonded to the base-cloth, enables VELROC casts to be applied simply and rapidly and they are more economical in use because of their strength and faster setting. VELROC casts are stronger, yet light and comfortable to wear, and do not separate into layers or become soggy. They cut crisply and cleanly and can be easily removed.

PRODUCTS TO MATCH
THE SURGEON'S SKILL

Johnson & Johnson
(Gt Britain) Ltd

HOSPITAL DIVISION SLOUGH BUCKS

* trade mark

JJ 4

"Let's face it....." *with a mug like yours,"* our Oldest Hand pointed out to the apprentice. *"you've got nothing to worry about. But the rest of us often need to protect our faces as well as our hands with a first class barrier cream!"*

"And what," muttered the lad to himself *"is going to protect me from him ...."*

STERNOL **Sternocleanse barrier creams**

There is a grade of Sternocleanse to suit your particular requirements.

* Are used throughout the works of the leading motor car and aircraft factories.
* Are the only Barrier Creams to contain Hexachlorophene bactericide.
* Have been awarded the Institute of Public Health & Hygiene Certificate.
* Are the results of 25 years experience in industry.
* Are suitable for the protection of the face, as well as hands and arms.

STERNOL LTD · ROYAL LONDON HOUSE · FINSBURY SQUARE · LONDON E.C.2. TEL: MONARCH 3871-5

THE WRIGHT PEAK FLOW METER

for the convenient and easy measurement of ventilatory capacity

Please direct Enquiries to

Airmed LIMITED

ONE OF THE CLEMENT CLARKE GROUP

Head Office
Edinburgh Way, Harlow, Essex

Home Sales:
16 Wigmore Street, W.1. Tel.: MUSeum 7037

VELROC*
plaster of paris bandages

Top of the bill with a strong supporting cast—VELROC Plaster of Paris Bandages. A strong, quicker setting, creamy plaster, firmly bonded to the base-cloth, enables VELROC casts to be applied simply and rapidly and they are more economical in use because of their strength and faster setting. VELROC casts are stronger, yet light and comfortable to wear, and do not separate into layers or become soggy. They cut crisply and cleanly and can be easily removed.

PRODUCTS TO MATCH
THE SURGEON'S SKILL

Johnson & Johnson
(Gt Britain) Ltd

HOSPITAL DIVISION SLOUGH BUCKS

* trade mark

JJ 4

"Let's face it....." *with a mug like yours,"* our Oldest Hand pointed out to the apprentice. *"you've got nothing to worry about. But the rest of us often need to protect our faces as well as our hands with a first class barrier cream!"*

"And what," muttered the lad to himself *"is going to protect me from him ...."*

STERNOL **Sternocleanse** barrier creams

There is a grade of Sternocleanse to suit your particular requirements.

* Are used throughout the works of the leading motor car and aircraft factories.
* Are the only Barrier Creams to contain Hexachlorophene bactericide.
* Have been awarded the Institute of Public Health & Hygiene Certificate.
* Are the results of 25 years experience in industry.
* Are suitable for the protection of the face, as well as hands and arms.

STERNOL LTD · ROYAL LONDON HOUSE · FINSBURY SQUARE · LONDON E.C.2. TEL: MONARCH 3871-5

THE WRIGHT PEAK FLOW METER

for the convenient and easy measurement of ventilatory capacity

Please direct Enquiries to

Airmed LIMITED

Head Office:
Edinburgh Way, Harlow, Essex

Home Sales:
16 Wigmore Street, W.1. Tel.: MUSeum 7037

ONE OF THE CLEMENT CLARKE GROUP

VELROC
plaster of paris bandages

Top of the bill with a strong supporting cast—VELROC Plaster of Paris Bandages. A strong, quicker setting, creamy plaster, firmly bonded to the base-cloth, enables VELROC casts to be applied simply and rapidly and they are more economical in use because of their strength and faster setting. VELROC casts are stronger, yet light and comfortable to wear, and do not separate into layers or become soggy. They cut crisply and cleanly and can be easily removed.

PRODUCTS TO MATCH
THE SURGEON'S SKILL

Johnson & Johnson
(G.B.) LTD.
HOSPITAL DIVISION · SLOUGH · BUCKS

"Let's face it....." *with a mug like yours,*" our Oldest Hand pointed out to the apprentice. "*you've got nothing to worry about. But the rest of us often need to protect our faces as well as our hands with a first class barrier cream!*"

"*And what,*" muttered the lad to himself "*is going to protect me from him . . . .*"

STERNOL **Sternocleanse** *barrier creams*

There is a grade of Sternocleanse to suit your particular requirements.

* Are used throughout the works of the leading motor car and aircraft factories.
* Are the only Barrier Creams to contain Hexachlorophene bactericide.
* Have been awarded the Institute of Public Health & Hygiene Certificate.
* Are the results of 25 years experience in industry.
* Are suitable for the protection of the face, as well as hands and arms.

STERNOL LTD · ROYAL LONDON HOUSE · FINSBURY SQUARE · LONDON E.C.2. TEL: MONARCH 3871-5

THE WRIGHT PEAK FLOW METER

for the convenient and easy measurement of ventilatory capacity

Please direct Enquiries to

Airmed LIMITED

ONE OF THE CLEMENT CLARKE GROUP

Head Office:
Edinburgh Way, Harlow, Essex

Home Sales:
16 Wigmore Street, W.1. Tel.: MUSeum 7037

BEFORE WORK—
the right barrier cream

There is a range of Rozalex Barrier Creams for use against a wide variety of harmful materials. Advice on the correct choice of barrier cream for any particular occupation is freely available.

Two-fold attack on **DERMATITIS**

AFTER WORK—
the right ointment

Use of an ointment after washing helps to keep the skin in good condition, and is recommended in the Ministry of Labour Form 330. Rozalex No. 4 Ointment helps to prevent chafing and cracking.

Safeguard working hands with ROZALEX

For further details and advice, write to:
ROZALEX LIMITED
(The Pioneers of Barrier Creams) 10 Norfolk Street, Manchester 2

TGA R157A

DIPLOMA IN INDUSTRIAL HEALTH
of the
SOCIETY OF APOTHECARIES OF LONDON

The Society of Apothecaries of London, conscious of the paramount importance of an efficient health service in Industry, has instituted this Diploma in Industrial Health, which is of a very high standard and is open to registered medical practitioners, members of H.M. Services, and others, such as Examining Surgeons, etc.

The Examination—which consists of three Papers, Oral and Clinical — is held in July and December.

The next Examination begins on December 4th, 1961.

Regulations and Forms of Application for Admission to the Examinations may be obtained from

The Registrar,
SOCIETY OF APOTHECARIES OF LONDON
BLACK FRIARS LANE, E.C.4

HOW MANY JOURNALS SHOULD YOU READ?

Once a man has applied himself to the practice of medicine he is in duty bound to keep himself informed on every advance in medical knowledge—and how can he do this except by reading?

The literature pours from the printing presses of the world, some of it of lasting significance, some ill-founded or misguided. How can a busy man pick his way through this maze?

ABSTRACTS OF WORLD MEDICINE does it for you . . .

This journal covers the whole field of clinical medicine and allied medical sciences in each issue, and presents to you each month concise, informative, readable abstracts of the best of the original papers published throughout the world—selected from over 1,500 medical journals.

ABSTRACTS OF WORLD MEDICINE saves your time . . .

Send your subscription now, or ask for further details:

Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1

Annual Subscription (12 issues) £4 4s. U.S.A. and Canada $13.50

PRINTED IN ENGLAND BY THE THANET PRESS, MARGATE